VOLUME 10 • NUMBER 6 • *June 1955*

Geriatrics

DEVOTED TO DISEASES AND PROCESSES OF AGING

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Allergic Skin Diseases 265

Aging of Musculoskeletal System 274

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Suicidal Attempts in Old Age 291

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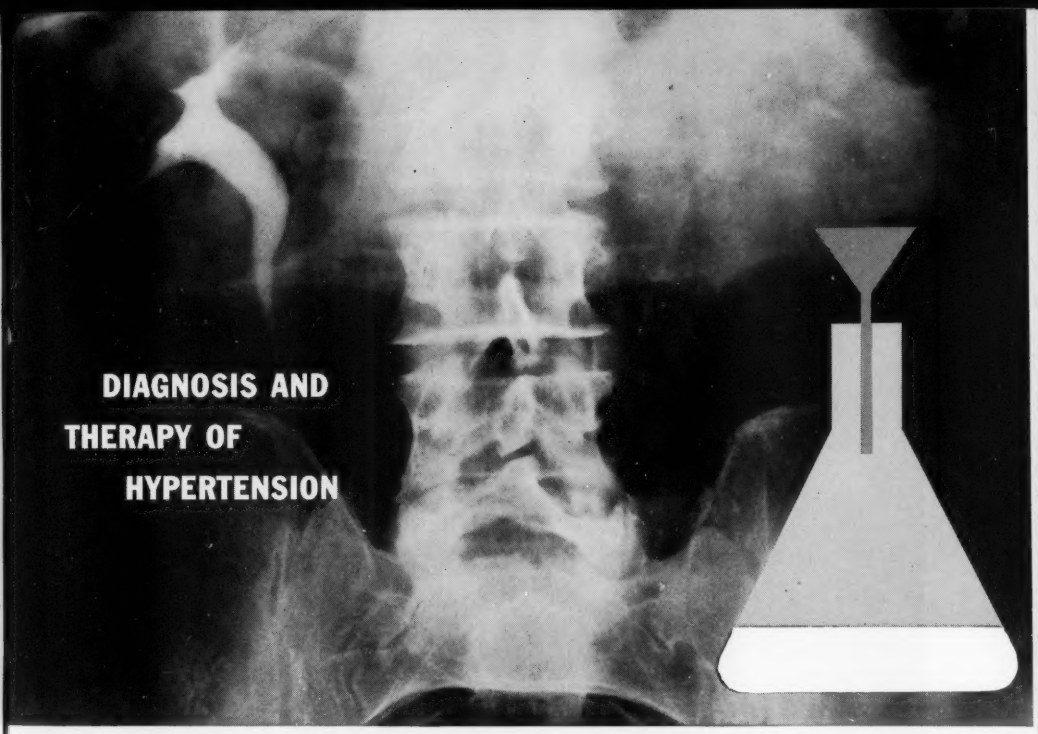

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*Dennis, E.; McConn, R. G.; Ford, R. V.; Hughes, W. M.; Beazley, H. L., and Moyer, J. H.: Postgrad. Med. *16*:300 (Oct.) 1954.

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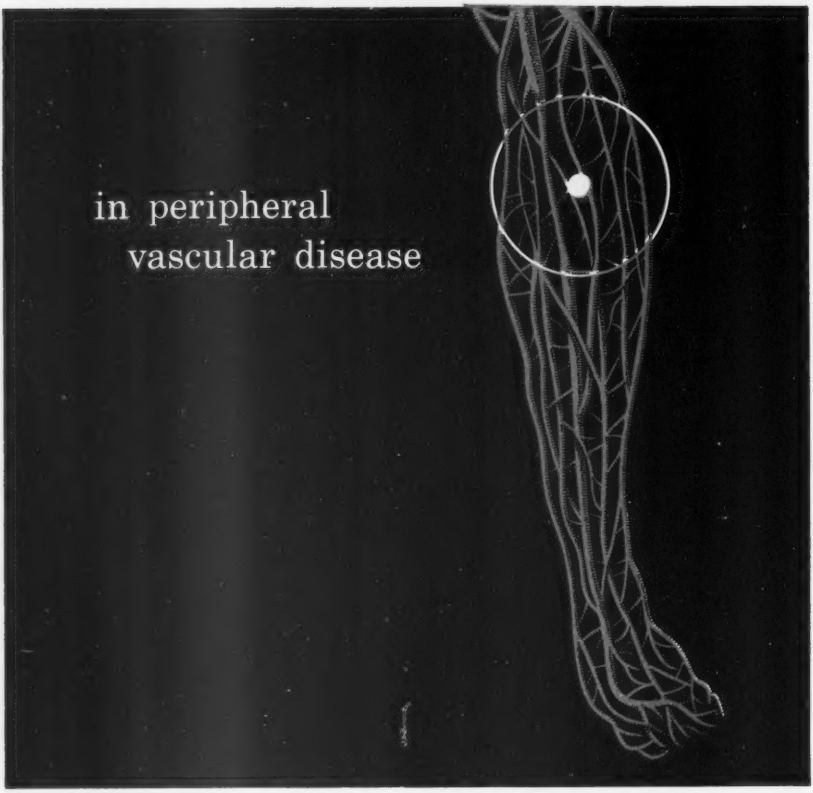

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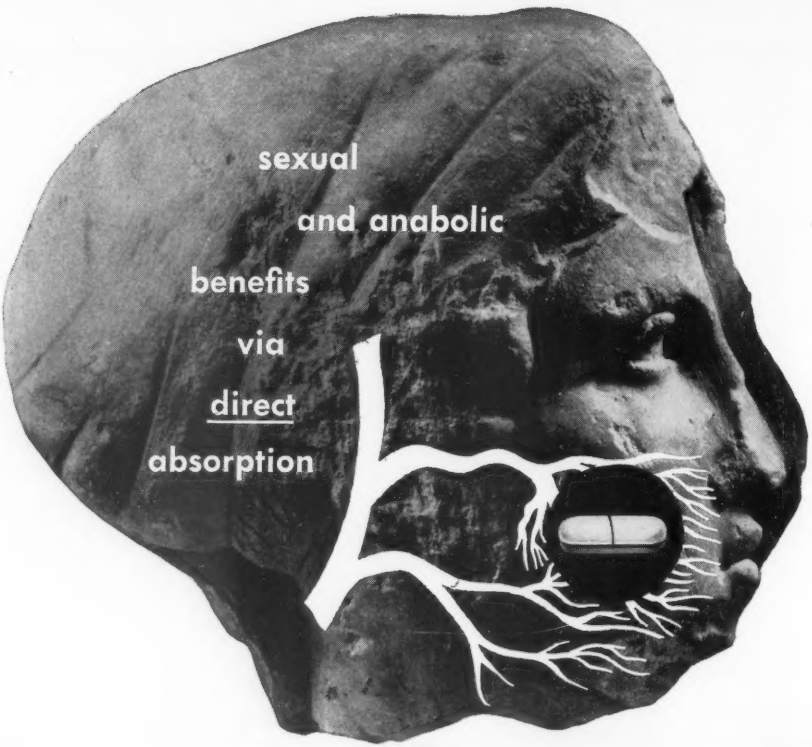

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Geriatrics

Established 1946

Volume 10
Number 6

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...looking forward

● Anticoagulant therapy is hazardous and should be undertaken only by physicians who have mastered the technic and who are willing to give the meticulous care that is required, warns William T. Foley, chief of the Vascular Clinic of New York Hospital, Cornell Medical Center, writing in the July issue of Geriatrics. In a critical review of *The Use of Anticoagulants in Geriatrics*, he gives results of his own extensive experience with anticoagulant drugs, and cites several case histories of patients, some of whom have been maintained as long as nine years on continuous dicumarol therapy.

● Focal cerebral lesions are difficult to recognize in the elderly, in whom the few signs which may be present may be overshadowed by changes in behavior and intellectual impairment, according to R. S. Allison, senior physician, Department of Neurology, Royal Victoria Hospital, Belfast, North Ireland. Two groups of patients, one with focal cerebral lesions and one with diffuse cortical atrophy, were compared in a study of the *Changes in Behavior and Impairment of Memory and Intellect in Later Life*. The general loss of intellectual function was found to be greater in the second group, as were the changes in mood and behavior, loss of memory for past events, and loss of insight.

● That mechanical factor plays an important pathogenic role in a great number of cases of *Localized Fibrositis* is the belief of Irvin Neufeld, associate in orthopedic surgery, New York Medical College. He discusses the various forms due to mechanical, traumatic, and therapeutic factors. Although cortisone, antiarthritics, and vitamin E have proved of little value in treatment, such measures as local procainization, ethylchloride spray, deep massage, and tonic and relaxing exercises, may be expected to bring gratifying results in many cases.

● Results of *Orbital Undercutting in the Aged* as applied to 20 elderly persons suffering from depression, anxiety, or obsession, are reported by William B. Scoville, chairman, Department of Neurosurgery, Hartford Hospital, Hartford, Connecticut, and W. Gerard Ryan, physician in charge, Elmcrest Manor, Portland, Connecticut. This method, which causes a minimum of personality blunting, was found to have gratifying results, which were unexpectedly superior to those in the younger age groups.

● *Rehabilitation of the Patient with Myocardial Infarction* is discussed by Jerome G. Kaufman and Marvin C. Becker of Newark, New Jersey. They believe that it is vitally important that these cardiac patients return to gainful employment as soon as feasible. Work records indicate that these patients make an excellent adjustment, are good workers, and have a low rate of absenteeism. Following early return to work, they are less prone to emotional instability. At present, the best way to judge a patient's work capacity is by careful, intelligent evaluation of the patient on the job by a psychiatrically oriented internist.

For these and other articles, abstracts, reviews, and special features, read every issue of Geriatrics.

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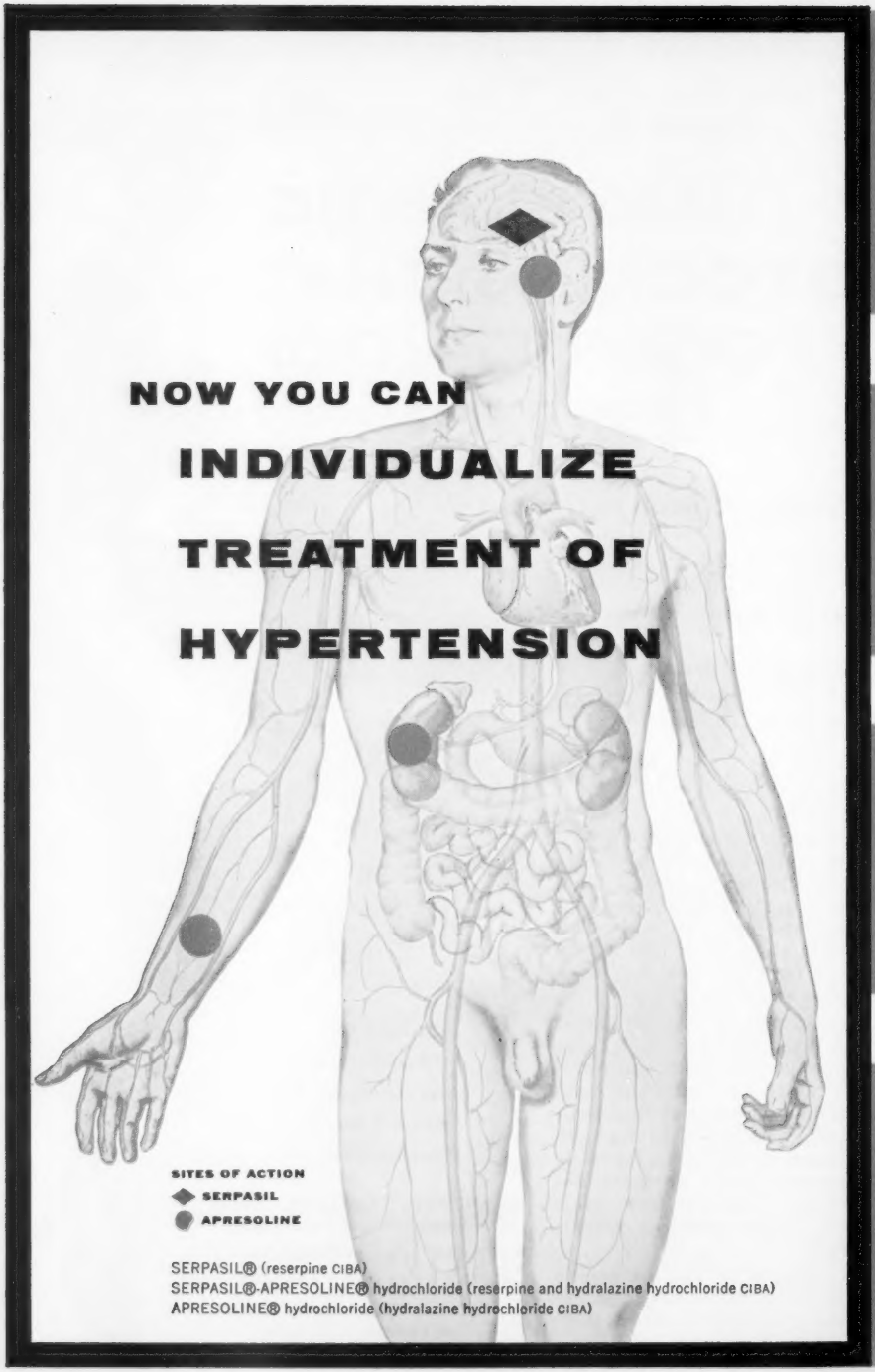
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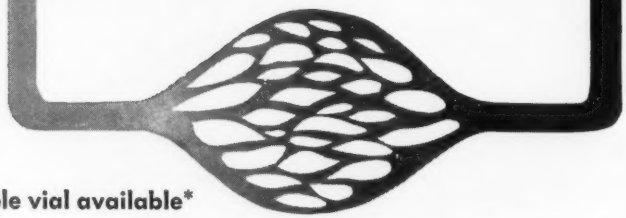

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1. Eisfelder, H. W.: Am. Pract. & Dig. Treat., 5:778 (Oct.) 1954.

2. Sebrell, W. H., Jr.: J.A.M.A., 152:42 (May) 1953.

3. Sherman, R. J., M.D.: Medical Times, 82:107 (Feb.) 1954.

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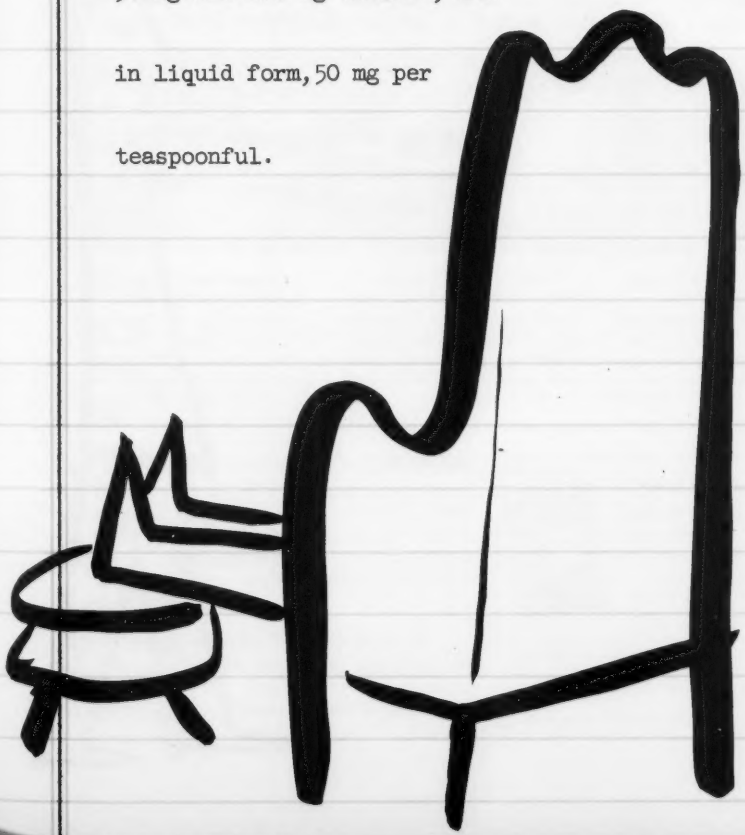

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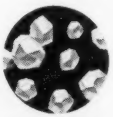

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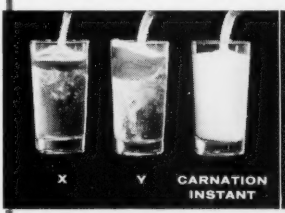

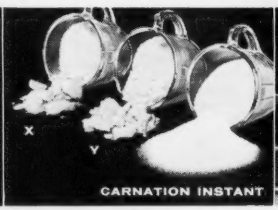

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1. Sadove et al.: J.A.M.A. *155*:626 (June 12) 1954.
2. Homburger, F., and Smithy, G.: New England J. Med. *251*:820 (Nov. 11) 1954.
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1. Palmer, H., and Gallagher, D. J. A.: Brit. M. J. *2*:558 (Sept. 2) 1950. 2. Ziegler, D. K., and Torres, F.: Neurology *5*:197 (March) 1955. 3. Timberlake, W. H., and Schwab, R. S.: New England J. Med. *247*:98 (July 17) 1952.

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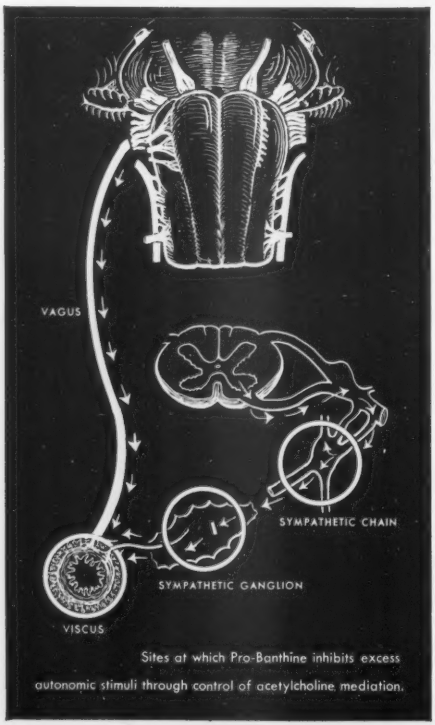

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1. Schwartz I. R.; Lehman, E.; Ostrove, R., and Seibel, J. M.: Gastroenterology *25*:416 (Nov.) 1953.

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1. Daly, J.W.: Am. J. M. Sc. *228*:440 (Oct.) 1954

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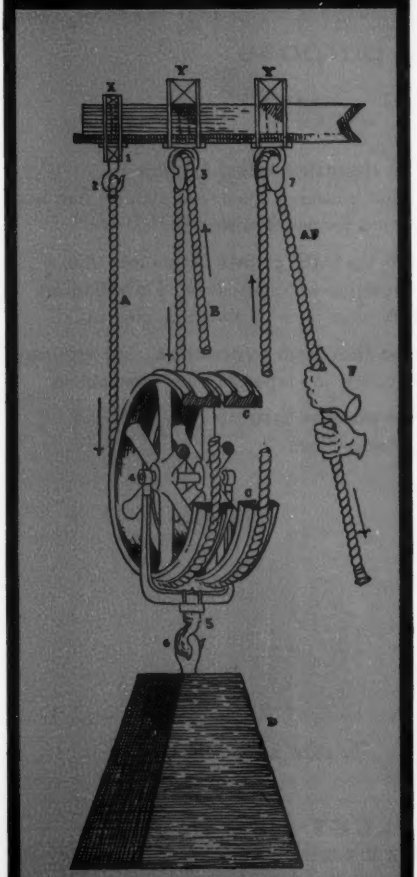

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Volume 10, Number 6 Geriatrics

Stress and disease

HANS SELYE, M.D.
MONTREAL, CANADA

The cumulative effect of everyday stress may become pathogenic and add to the premature development of senility. Study was made of the precise pathways through which stress acts upon the various parts of the body and the findings correlated with observations from the world literature. The essential facts are summarized in a brief synoptic review for gerontologists.

◆ Stress and the so-called "diseases of adaptation," which are due principally to an imperfect adjustment to stress, play an especially prominent role in geriatrics, since the pathogenic actions of exposure tend to be cumulative and become most evident during the later years of life. In contrast to the specific remedies—vitamins, antibiotics, chemotherapeutic agents, and hormones substituting for a destroyed endocrine gland—used against specific maladies, both the cause and the treatment of stress diseases are essentially *nonspecific*.

Nonspecific therapy, such as injection of foreign proteins, use of colloidal metals, exposure to heat or cold, and shock therapy, has been practiced for many centuries, but its progress has been slow as compared with that of specific therapy. The principal reason for this is that its development had to be guided almost solely by empiricism. There were no means available for the analytic dissection of the mechanism of nonspecific reactions into its ingredient parts. Only during the last twenty years has it become possible to identify certain pathways through which nonspecific defense reactions against stress are mediated.

The most striking progress was made in elucidation of the role of the anterior pituitary and the adrenal cortex in resistance to stress and in the genesis of the mesenchymal diseases. Until a few months ago, the stress-disease concept still lacked proof of one of its fundamental postulates.

One of these basic tenets is that, under the influence of various types of exposure, the adrenal cortex produces two kinds of corticoids which regulate

HANS SELYE *is professor and director of the Institute of Experimental Medicine and Surgery at the University of Montreal.*

mesenchymal reactions: (1) the cortisone type, consisting of those which inhibit inflammation, cause lymphatic involution, and induce catabolism of the body as a whole, and (2) the desoxycorticosterone type, consisting of those which appease all these effects. Until quite recently, however, some of the outstanding specialists in the field of adrenal physiology could not accept this tenet for they doubted that the adrenal could secrete desoxycorticosterone or any similar cortisone-antagonizing hormone. This unitarian theory of cortical function was disproved recently by the discovery of a new corticoid, aldosterone, the presence of which could be ascertained not only in the adrenals but in circulating blood. Only a few months ago, we succeeded in demonstrating that this new substance, with a close chemical relationship to desoxycorticosterone, does antagonize cortisone-like compounds. Thus, the theory of a balance between two opposing types of corticoids has been verified by direct observation.[1]

I should like to present a brief synopsis of our work on stress as an aid in appraising the manifold relationship between this rapidly growing field of medical research and geriatrics. The entire world literature dealing with such maladies as cardiovascular disease, nephrosclerosis, metabolic diseases of the aged, and osteoporosis from the viewpoint of stress and the adaptive hormones, has been surveyed in two monographs[2,3] and a series of annual reports.[4-6,1] Here we shall restrict ourselves to the principal observations upon which the stress concept rests.

Precursors of the Stress Concept

Ever since man first used the word disease he has had some inkling of the stress concept. The fact that a single term can be used to denote a great variety of individual maladies indicates that they have something in common—that they possess some nonspecific features which permit us to distinguish disease from the condition of health. Yet, precisely because these manifestations are not characteristic of any one disease, they have received little attention in comparison with the specific ones. They were thought to be of lesser interest for, unlike the specific characteristics, they did not help in recognition of the eliciting pathogen nor lend themselves to any specific therapy.

Nevertheless, several early investigators attempted to clarify the mechanisms involved in such nonspecific reactions. Since neurology antedates endocrinology, it is understandable that, of the two great integrating systems, the nervous and the hormonal, the nervous system was the first to be examined from this point of view. Ricker, Speransky, Reilly, Hoff, and many others gathered important data on the part of the nervous system in such nonspecific reactions as fever, polymorphonuclear leukocytosis, and inflammation. Cannon's studies helped in understanding the role of the sympathetic nervous system and its humoral effector substances. Quite independently, much progress was made in the study of pituitary and adrenocortical hormones by chemists, physiologists, and clinicians.

Additional experiments were necessary, however, to show that the many nonspecific responses of individual target organs are closely integrated and represent part of a single biologic response, the general adaptation syndrome. These investigations made it evident that the stress pattern of reaction plays an integral part in the most varied physiologic, pathologic, and pharmacologic phenomena.

The Concept of Stress

A series of experiments on animals in 1936 showed that the organism responds in a stereotypical manner to widely different factors, such as infections, intoxi-

cations, trauma, nervous strain, heat, cold, muscular fatigue, and x-ray irradiation. The specific actions of all these agents are quite different. Their only common feature is that they place the body in a state of systemic stress. Hence, it was concluded that the stereotypical response, which is superimposed upon all specific effects, represents the somatic manifestations of nonspecific stress.

But what is nonspecific stress? The term had long been used in physics to denote the interaction between a force and the resistance opposed to it. For instance, pressure and tension can put inanimate matter under stress. The nonspecific response was thought to represent the biologic equivalent of such physical stress. The term has now been generally accepted in this sense not only in English, but—since attempts to translate "stress" led to much confusion—also in most other languages.

The General Adaptation Syndrome

The most outstanding manifestations of the stress response were adrenocortical enlargement with histologic signs of hyperactivity; thymicolymphatic involution with certain concomitant changes in the blood count such as eosinopenia, lymphopenia, and polynucleosis; and gastrointestinal ulcers, often accompanied by other signs of damage or shock.

Although all the organs of the body showed involutional or degenerative changes, the adrenal cortex actually seemed to flourish on stress. We suspected that this adrenal response had a useful part in the systemic, nonspecific adaptive reaction, which was visualized as a call to arms of the body's defense forces and named the alarm reaction.[7]

Later studies showed that the alarm reaction is but the first stage of a much more prolonged general adaptation syndrome, known as G-A-S. This syndrome comprises three distinct stages: (1) *alarm reaction* (A-R), in which adaptation has not yet been acquired; (2) *stage of resistance* (S-R), in which adaptation is optimal; and (3) *stage of exhaustion* (S-E), in which the acquired adaptation is lost again.

The Mechanism of the General Adaptation Syndrome

A study in which rats were adrenalectomized and then exposed to stressor agents, showed that, in the absence of the adrenals, stress can no longer cause thymicolymphatic involution or characteristic blood count changes.[8] When adrenalectomized rats were treated with the impure cortical extracts then available, it became evident that thymicolymphatic involution and the typical blood count changes could be produced by adrenal hormones even in the absence of the adrenals. Therefore, these changes were considered to be indirect results of stress mediated by corticoids.[8,9]

Conversely, gastrointestinal ulcers and other manifestations of pure damage or shock were more severe in adrenalectomized than in intact animals and could be lessened by treatment with cortical extracts. It was concluded that these lesions are not mediated through the adrenal but are combated by an adequate adrenocortical response to stressor agents.[8] Since, among many surgical interventions tried, only hypophysectomy prevented adrenal response during the alarm reaction, it was concluded that stress stimulates the cortex through an adrenocorticotropic hormone, now known as ACTH.[10, 11]

When pure cortical steroids became available, it was shown that administration of *mineralocorticoids* or M-Cs, such as desoxycorticosterone, produces experimental replicas of the so-called hypertensive and inflammatory rheumatic diseases – notably, nephrosclerosis, hypertension, and vascular lesions[12]—as well as arthritic changes resembling, in acute experiments, those of rheumatic fever and, after chronic treatment, those of rheumatoid arthritis.[13] Yet, even high

doses of mineralocorticoids did not induce any noteworthy thymicolymphatic or blood count changes, such as were caused by cortical extracts.

Significantly, exposure of animals to certain nonspecific stressor agents, such as cold, produced adrenocortical enlargement and organ changes similar to those elicited by the administration of mineralocorticoids.[14] The unitarian theory, which held that the adrenal gland secretes only one corticoid, so that a derangement in the balance between antagonistic cortical hormones is impossible, was disproved by the isolation of aldosterone from both the tissue and venous blood of the adrenals.[15]

On the other hand, extracts rich in glucocorticoids, or G-Cs, such as cortisol and cortisone, were potent in causing thymicolymphatic involution and in eliciting the characteristic blood count changes of the alarm reaction. They also tended to inhibit the inflammatory, rheumatic-like changes which can be elicited in animals by mineralocorticoids. Thus, in many respects, the two types of corticoid hormones antagonize each other.[9, 2]

Another interesting activity of the corticoids, discovered at about this time, is their singular effect upon the central nervous system of animals. A variety of steroids, including G-Cs, M-Cs, and other steroid hormones, and hormone-metabolites such as pregnanediol and pregnanedione, were shown to have the property of causing great excitation and confusion, followed by depression of all reflex activities and, eventually, deep anesthesia.[16] This raised the question whether a pronounced increase in the activity of endogenous corticoids could be responsible for certain nervous and emotional accompaniments of exposure to stress. After the introduction of cortisone into clinical therapy, it became evident that, in man, this hormone can also exert a powerful effect upon the central nervous system. In animals, both G-Cs and M-Cs exhibit this effect, hence we shall have to watch for it as soon as patients are treated with large doses of aldosterone.

The terms glucocorticoids and mineralocorticoids emphasize the salient metabolic actions of these substances; clinically, their effects upon inflammation are of even greater interest. Since the glucocorticoids inhibit inflammation, and the mineralocorticoids enhance it, the G-Cs may appropriately be called *antiphlogistic corticoids* or A-Cs and the M-Cs, *prophlogistic corticoids* or P-Cs, when they are discussed with reference to their effects upon inflammation. It remains to be seen, however, whether G-C and A-C, or M-C and P-C, activities run parallel in all steroid compounds, including those which, like aldosterone, have not yet been fully examined for these effects.

Inflammatory granulomas, especially those produced near joints by local application of irritants such as formalin and mustard powder, as well as certain allergic reactions, are also aggravated by P-Cs and inhibited by A-Cs. Apparently, the response of the adrenal cortex is important not only in reaction to systemic stress, but also in the many topical defense reactions which occur upon exposure to local stress.[17, 1] These findings helped to formulate the concept of the local adaptation syndrome, or L-A-S, to be discussed later.

Our attention was first called to a possible relationship between the adrenal cortex and inflammation by an incidental observation on rats. It was found that the rat is naturally hypersensitive to egg white, and responds to intraperitoneal or intravenous administration of this substance by an acute serous inflammation of paws and snout. This response was aggravated in adrenalectomized animals, presumably because they could not defend themselves by the endogenous production of A-Cs. On the other hand, it was prevented by treatment with systemic stressors directly in proportion to the adrenocortical enlargement they pro-

duced, presumably as a result of excess A-C secretion.[11] It was later shown that cortisone and ACTH inhibit, while certain crude anterior-pituitary preparations and desoxycorticosterone aggravate this *anaphylactoid* type of acute inflammation.[18]

Curiously, our *crude anterior pituitary extracts* also duplicated most of these P-C actions upon the cardiovascular system, blood pressure, connective tissue, and kidneys.[19, 9] The hypophysial preparations which were used were corticotropic in that they enlarged the adrenal cortex, but they were particularly rich in *somatotropic*, or *growth, hormone* (STH). This made it difficult to interpret our early experiments, in which crude extracts were used, because we could not distinguish clearly between effects of ACTH and STH. With purified ACTH, however, it became evident that the pathogenic actions of the crude anterior pituitary preparations were not due to their ACTH content, since even the highest tolerable doses of the pure corticotropic hormone failed to duplicate their predominant P-C effects. On the other hand, overdosage with purified STH caused cardiovascular and renal lesions virtually identical with those previously observed in animals treated with P-Cs. It was concluded that the characteristic actions of our crude anterior-pituitary preparations were probably due to their STH content. It is still undetermined to what extent STH acts indirectly by stimulating P-C production of the adrenal cortex, or directly by sensitizing peripheral tissues to P-Cs, although preliminary observations suggest that the latter mechanism is more important.[20]

The most interesting role of STH from the internist's viewpoint is that it can combat catabolism and susceptibility to infections. Animals overdosed with ACTH or A-Cs lose a great deal of weight and eventually die, almost always from generalized septicemia, caused by normally saprophytic microorganisms. In rats, the lung tissue appears to be singularly predisposed to such infections. Under these conditions, adequate doses of STH prevent weight loss as well as excessive microbial proliferation.[21] Whether STH will prove valuable in the management of infections in man is undetermined, but experiments on rats have demonstrated the influence of adaptive hormones upon resistance to the human type of tuberculosis. The rat, normally resistant to tuberculosis bacilli, may be sensitized by ACTH or A-Cs, and this sensitivity can in turn be abolished by STH.[22, 23]

Conditioning of Hormone Actions

As work progressed, it became more obvious that the activity of the stress hormones depends largely upon a variety of conditioning factors, such as heredity, age, previous exposure to stress, and the nutritional state. Thus, production of corticotropic hormone by the pituitary is enhanced by a high-protein diet, while the action of M-Cs upon most target organs is augmented by excess sodium.[9]

Stress itself is the most effective factor in conditioning actions of adaptive hormones. Systemic stress augments the antiphlogistic, lympholytic, catabolic, and hyperglycemic actions of A-Cs, but the adaptive hormones cannot act in modifying the course of inflammation, unless some topical stressor first elicits a phlogistic response.

Ingle[24] introduced the concept of the "permissive actions" of corticoids, in which the adrenal hormone does not affect a target of stress, although it permits a stressor to act upon it. Furthermore, the presence or absence of a permissive factor can only allow or disallow a reaction, but cannot vary its intensity. To illustrate, the production of light by an electric lamp might be compared to the biologic reaction and the switch to the permissive factor. The switch cannot produce light nor regulate the degree of its intensity, but unless it is turned on,

the lamp will not function. Correspondingly, the functional signs—thought to be characteristic of corticoid overproduction during stress—would not result from actual increase in corticoid secretion, but from the extra adrenal actions of the stressors. Corticoids would be necessary only in a supporting capacity to maintain vitality and reactivity of tissues.

Actually, it is in the specific and not in the nonspecific stress reactions that the corticoids play a purely permissive role of this type. Here they are necessary only to prevent stress and collapse, thus keeping the tissues responsive. For instance, adrenalectomized rats will not respond to injected STH with somatic growth, or to sexual stimulation with mating, without a minimal maintenance corticoid dosage. These reactions are not characteristic of the corticoids and cannot be duplicated in the absence of the specific stimulus, even with the highest doses of corticoids. The characteristic signs of A-C overproduction seen in the alarm reaction—atrophy of the lymphatic organs, catabolism, and inhibition of inflammation—are also impeded by adrenalectomy and restored by maintenance doses of A-Cs in the presence of stress, which sensitizes the tissues to them. However, unlike specific actions, these nonspecific effects can also be duplicated in the absence of any stressor if large doses of A-Cs are given.[1]

These conditioning influences are particularly important in the regulation of stress reactions, because, in the final analysis, they can determine whether exposure to a stressor will be met by a physiologic adaptation syndrome, or cause diseases of adaptation. Furthermore, they can determine the selective breakdown of the different organs. Differences in predisposition due to such factors may explain why the same kind of stressor can cause various types of diseases of adaptation in different individuals.

It was only on the basis of such experiments that the mechanism through which stress affects the adrenal cortex could be explained. Stressors such as formaldehyde remained without effect upon the adrenal, even if the adrenal cortex was kept from atrophy by administration of pituitary extract. The effect of such a drug on the adrenal appears to be an indirect one due to pituitary stimulation.[10]

The Local Adaptation Syndrome

It had long been known that many local responses to injury are nonspecific—that a variety of topical stressors such as burns, microbes, and drugs share the power of producing nonspecific tissue damage and inflammation. Only recently has the close relationship between systemic and local types of nonspecific reactions been clarified. While the characteristic response of the body to systemic stress is the general adaptation syndrome, topical stress elicits a local adaptation syndrome (L-A-S), whose principal repercussions are confined to the immediate vicinity of the eliciting injury. They consist, on the one hand, of degeneration, atrophy, and necrosis, and on the other of inflammation, hypertrophy, hyperplasia, and, under certain conditions, neoplasia.[1]

L-A-S and G-A-S

At first sight, there appears to be no great similarity between the systemic and the local reaction types. A patient in traumatic shock furnishes a characteristic example of the G-A-S, particularly of its earliest stage, the shock phase. An abscess formed around a splinter of wood represents the L-A-S and particularly its stage of resistance, during which defensive inflammatory phenomena predominate. These two instances of disease reveal no surface likenesses, yet more careful study shows them to be closely related.

The experimental observations which led to these conclusions have been re-

ported.[25, 26] The G-A-S and the L-A-S are thought to be interrelated because:

1. Both are nonspecific reactions, comprising damage and defense;
2. Both are triphasic, with typical signs of crossed resistance or, depending upon the stressors used, crossed sensitization during the second stage;
3. Both are singularly sensitive to the so-called adaptive hormones;
4. If the two reactions develop simultaneously in the same individual, they greatly influence one another—that is, systemic stress alters tissue reactivity to local stress and vice versa.

The Diseases of Adaptation

It was concluded that the pathogenicity of many systemic and local stressor agents depends largely upon the function of the hypophyseal-adrenocortical system. The latter may either enhance or inhibit the body's defense reactions against stressors. Derailments of this adaptive mechanism are the principal factors in the production of certain maladies which are considered to be essentially diseases of adaptation.

Such diseases do not necessarily become manifest during exposure to stress. This is demonstrated by the fact that temporary overdosage with desoxycorticosterone can initiate a self-sustaining hypertension, which eventually leads to death, long after hormone administration has been discontinued. Here, we speak of "metacorticoid" lesions. The possibility that a temporary excess of endogenous aldosterone can induce similar delayed maladies deserves serious consideration.

Among the derailments of the G-A-S which may cause disease, are the following:

1. An absolute excess or deficiency in the amount of adaptive hormones produced during stress.
2. An absolute excess or deficiency in the amount of adaptive hormones retained, or fixed, by their peripheral target organs during stress.
3. A disproportion in the relative secretion or fixation during stress of various antagonistic adaptive hormones—ACTH and A-Cs, on the one hand, and of STH and P-Cs, on the other.
4. Production by stress of metabolic derangements, which abnormally alter the target organ's response to adaptive hormones through the phenomenon of conditioning.
5. Although the hypophysial-adrenal mechanism plays a prominent role in the G-A-S, other organs which participate in the syndrome may also respond abnormally and become pathogenic during adaptation to stress.

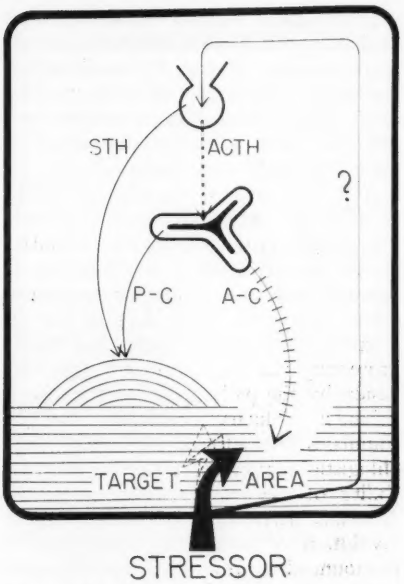

Fig. 1. The pathways of stress reaction. The stressor acts upon the target directly (thick arrow) and indirectly through the pituitary and adrenal. Through some unknown pathway the first mediator travels from the injured target area to the anterior pituitary, which discharges ACTH.

Conclusions

All agents which act upon the body or any of its parts exert dual effects: (1) specific actions, which modify the nonspecific actions of the same agents, and (2) nonspecific or stressor effects, whose principal pathways are illustrated in figure I.

It is quite possible that this first mediator of hormonal defense is not always the same. It may be an adrenaline discharge, a liberation of histamine-like toxic tissue metabolites, a nervous impulse, or even a sudden deficiency in some vital body constituent.

ACTH stimulates the adrenal cortex to discharge corticoids, some of which, the prophlogistic corticoids, stimulate the proliferative ability and reactivity of connective tissue and enhance the inflammatory potential. They help to put up a strong barricade of connective tissue, protecting the body against further invasion by the pathogenic stressor agent.

Under ordinary conditions, ACTH stimulates the adrenal to secrete antiphlogistic corticoids, which inhibit the ability of the body to put up granulomatous barricades and tend to cause involution of connective tissue with a pronounced depression of the inflammatory potential. Thus they open the way to spread of infection.

It is not yet known whether ACTH always stimulates the adrenal to produce the various corticoids in the same proportions and with a predominance of A-Cs. Recent experiments suggest that, depending upon conditions, ACTH may cause the predominant secretion of one or the other of the steroid hormones.[27] However, the somatotropic hormone of the pituitary increases the inflammatory potential of connective tissue and sensitizes the target area to the actions of the P-Cs.

Possibly the hypophysis also secretes a special corticotropin which induces the adrenal to elaborate predominantly P-Cs. The somatotropic hormone itself may possess such effects, but this has not been proved. Even if ACTH were the only corticotropin, the actions of the corticoids produced under its influence can be vastly different, depending upon conditioning factors such as STH, which specifically sensitize the target area for one or the other type of corticoid action. Actually, conditioning factors could alter the response of the adrenal cortex to ACTH, so that its cells would produce more A-Cs or P-Cs. Thus, during stress, one type of effect predominates.

The fundamental reaction pattern to topical stressors is a local adaptation syndrome with inflammation; to systemic stressors, the general adaptation syndrome. Various modifications of these two basic responses constitute the essence of most diseases.

Predictions

Pasteur, Koch, and their contemporaries introduced the concept of specificity into medicine. Each individual, well-defined disease, they held, has its own specific cause. It has been claimed that Pasteur, too occupied with the pathogen, failed to recognize the importance of the terrain, but his work on induced immunity shows that this is incorrect.

The theory which was followed by Pasteur and his successors in their most fruitful investigations held that the organism can develop specific adaptive reactions against individual pathogens and that by imitating and complementing these, whenever they are short of optimal, we can treat many of the diseases which are due to specific pathogens.

The G-A-S represents, in a sense, the negative counterpart, or mirror image, of this concept. It holds that many diseases have no single cause, no specific pathogen, but are due largely to nonspecific stress and to pathogenic situations resulting from inappropriate responses to such nonspecific stress.

Our blueprint of the pathways through which stress acts may be partly incorrect; it is certainly quite incomplete. But in it we have a basis for the objective scientific dissection of such time-honored but vague concepts as "reactivity," "constitution and resistance," and "nonspecific therapy" in the genesis and treatment of disease.

We should like to reiterate that, in our

opinion, research on stress will be most fruitful if it is guided by the theory that we must learn to imitate—and if necessary to correct and complement—the body's own autopharmacologic efforts to combat the stress factor in disease.

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Diagnosis of polyps of the rectum and colon

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◆ More frequent use of the sigmoidoscope and improved methods of x-ray study have greatly enlarged our knowledge of the clinical course of polyps of the rectum and colon. It should be emphasized that

1. Rectal bleeding in patients of 50 or more is the most common symptom of a polyp in the lower intestinal tract.
2. All patients with rectal bleeding should have sigmoidoscopic examination of the rectum and x-ray studies of the colon.
3. For the purpose of diagnosis, the colon and rectum should be considered a single segment of the gastrointestinal tract.

Patients in the older age group who have rectal bleeding must be examined by the sigmoidoscope and barium enema and air contrast studies of the colon for the presence of polyps. The percentage of correct diagnoses will depend on the thoroughness of these two methods of examination, recognizing certain pitfalls in use of both.

Pathology

The common polyp of the colon and rectum has its origin in the mucosal elements of the bowel, and, since it contains both columnar epithelium and glandular epithelium on microscopic study, is classified as an adenoma. For clinical purposes, these adenomas can be divided into the peduncular or polypoid adenoma and the villous or flat adenoma on the basis of their gross appearance.

Polypoid adenoma, which comprises 95 per cent of all adenomas, is the benign tumor growing into the lumen of the bowel, with a stalk attached to the mucosa by a narrow base. The villous adenoma, which comprises 5 per cent of all adenomas, is a single flat tumor with papillary projections on the surface growing along the wall of the bowel and attached by a broad base. The polypoid adenoma occurs more frequently in the sigmoid and rectum and is often multiple. Grossly, it varies in appearance from the small sessile growth with little differentiation between base and stalk to the pedunculated growth with a large tumor attached to the mucosa by a long stalk.

The pedunculated or polypoid adenoma is more accessible to complete removal with a snare or wire loop. The villous adenoma, although microscopically benign, is clinically malignant because the margins of the lesion are poorly defined and its broad base makes complete removal difficult. The recurrence rate is high and carcinoma in situ is a frequent finding.

There are those who believe that both types of adenoma have a common origin from the mucosa and the variation in the gross appearance is one of growth variation.[1] This opinion arises from the microscopic finding of columnar and glandular epithelium in both villous and papillary adenoma. Dukes[2] in London believes that the growth variation between the two

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types is the result of a difference in origin. The ordinary polyp begins in the glandular epithelium in the mucosa, and in the process of growth and expansion protrudes into the lumen where it is influenced by intestinal peristalsis and the passing fecal stream and develops a stalk. The villous adenoma has its origin in the columnar epithelium of the lining mucosa, grows in a linear direction, is not subjected to the fecal stream, and has no stalk but a broad base. The relationship between polyps of the colon and rectum and malignancy is best stated by the observation that in those cases in which there is more than one polyp carcinomatous degeneration can be expected.

Diagnosis

Diagnosis of polyps of the colon and rectum is best made with the use of a sigmoidoscope, barium enema and air contrast studies of the colon, and a certain mental attitude toward the significance of rectal bleeding. Patients past 50 who report rectal bleeding are potential candidates for the diagnosis of a polyp. During a recent survey, polyps were found in 17 per cent of patients in the upper age bracket who presented no symptoms but who had roentgenograms made of the colon.[3] These figures correspond with those published in other series in which polyps were demonstrated by roentgenogram in the absence of rectal bleeding. Of all patients who undergo sigmoidoscopy during routine examination, 5.8 per cent will present one or more polyps in the rectum.

Blood in the rectum and the presence of a "sentinel" polyp are the two most significant findings on sigmoidoscopy. In either instance, the colon must be examined by roentgenogram. There are two conditions that may interfere with x-ray visualization of the colon – incomplete cleansing with fecal particles remaining in the bowel producing an x-ray finding similar to a polyp and distortion of the sigmoid colon by many diverticula. If either of these irregularities occurs, the x-ray studies must be repeated.

It is important in the search for polyps that lateral x-ray views of the sigmoid, splenic, and hepatic flexures be made because of the overlapping of the bowel in these areas. The sigmoidoscope is again used at the time of surgery to examine the bowel above and below the opening made for the removal of polyps. Polyps that are left in the colon after surgery are a potential source of carcinoma.

Case Reports

The following cases are taken from a series studied at the Los Angeles County General Hospital and in private practice to illustrate some of the pitfalls in the diagnosis and treatment of polyps of the colon and rectum.

Case 1. A 56-year-old woman was operated upon in 1951 for a polyp, at which time a single adenoma was removed from the sigmoid through a colotomy. Diagnosis was made preoperatively by sigmoidoscopy on the basis of rectal bleeding and the location of the lesion was demonstrated with a barium-enema x-ray study. Symptoms recurred and in 1953 an air contrast study showed multiple polyps in the sigmoid and transverse colon. A subtotal left colectomy was performed and carcinomatous degeneration was found in two of the polyps removed at operation.

Case 2. A 75-year-old woman presented symptoms of intermittent diarrhea, abdominal cramping, and occasional blood in the stool. Sigmoidoscopy was negative except for the presence of blood. A barium enema showed a constricting lesion of the transverse colon which was interpreted as a malignant lesion. An air contrast study of the colon was made showing the lesion of the transverse colon and a polyp in the cecum. A right colectomy was performed and the surgical specimen showed a constricting carcinoma of the transverse colon, two benign adenomas in the cecum, and one in the ascending colon.

Case 3. D. R., a 52-year-old woman, underwent surgery at another hospital in 1950 for a carcinoma of the sigmoid. The patient had a recurrence of bleeding by rectum in 1952. Sigmoidoscopy and x-ray studies of the colon failed to reveal any possible source of bleeding. In 1953 x-ray studies were repeated and a carcinomatous lesion of the ascending colon was demonstrated. Review of the x-ray studies of the colon made in 1952 showed a polyp that had been overlooked

at the site of the present carcinomatous lesion. These findings were confirmed at surgery.

Case 4. A 52-year-old woman had an operation at another hospital for a single polyp which was removed through an opening in the sigmoid colon. One year later a recurrence of rectal bleeding caused the patient to return to her surgeon. At this time a barium enema and air contrast studies of the colon showed two polyps, one in the sigmoid colon and one in the descending colon. These two lesions were removed at surgery.

Case 5. A 54-year-old man was admitted to the Veterans Administration Hospital at Sawtelle, California, with a history of rectal bleeding. Sigmoidoscopic studies and barium enema and air contrast studies showed a single polyp in the splenic flexure of the colon. At the time of surgery, an opening was made in the splenic flexure and a single polyp removed. A sigmoidoscope was introduced into the colon and the bowel examined to the middle third of the transverse colon and to the sigmoid flexure. Another polyp at the proximal third of the sigmoid was seen and removed through a second colotomy opening.

Case 6. A 57-year-old woman was hospitalized at St. Vincent's Hospital with clinical findings and a history suggestive of chronic intestinal obstruction. The history revealed that two years previously she had had a resection of the middle third of the sigmoid because of the presence of three polyps. These previous operative findings were confirmed by conversation with the surgeon. Sigmoidoscopy was negative. X-ray studies of the colon showed a constriction of the sigmoid at the point of previous anastomosis, with findings of impending complete obstruction. The patient was prepared for surgery and at operation the segment of obstructed sigmoid was resected. This segment showed an adenocarcinoma at the point of a cicatricial stenosis and two benign adenomas, one below and one above the point of constriction.

Case 7. E. W., a 60-year-old male physician, was admitted to St. Vincent's Hospital November 16, 1949, for resection of the middle third of the sigmoid for multiple polyps. Symptoms had begun nine months previously with intermittent rectal bleeding. At that time sigmoidoscopic examination revealed internal and external hemorrhoids. Barium enema study demonstrated a negative colon. Hemorrhoidectomy was performed at another hospital. The bleeding continued after the operation. Sigmoidoscopy performed before his second hospital admission was negative and air contrast study of the colon demonstrated two polyps in the sigmoid. At operation the resected sigmoid was found to contain one large polyp and two smaller ones. The patient has had no bleeding since the last operation.

Case 8. A 67-year-old man had a history of intermittent rectal bleeding. Sigmoidoscopy was negative for bleeding and polyps. An air contrast study of the colon was negative in the conventional anterior-posterior position. The patient was turned over and a single polyp was demonstrated in the posterior-anterior position. The polyp was removed at surgery. Operative sigmoidoscopy did not reveal other polyps in the colon.

Case 9. B. C., a 62-year-old man, was admitted to the Veterans Administration Hospital with various symptoms including occasional rectal bleeding. Sigmoidoscopy revealed internal and external hemorrhoids. Barium enema and air contrast studies of the colon demonstrated a polyp in the middle third of the sigmoid. The middle third of the sigmoid was resected and the pathologic report was that of polyp with adenocarcinoma grade II. The lymph nodes in the surgical specimen were negative for metastasis.

Case 10. A 52-year-old man was seen in the office with complaint of irregular and occasional rectal bleeding. Sigmoidoscopic examination was negative except for several external hemorrhoids. Air contrast study of the colon showed a defect at the junction of the descending and sigmoid colon. Since a stalk could not be demonstrated the roentgenograms of the colon were repeated and the defect, which was probably fecal debris, had disappeared. This patient should have repeat roentgenograms of the colon in three months.

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Selected allergic skin diseases in older persons

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Allergic dermatoses are somewhat less common in older than in younger people. The three most common allergic eruptions in persons over 50 are allergic eczematous contact dermatitis, allergic drug eruptions, and allergic urticaria. Topically and systemically administered medicaments are among the most frequent causes of allergic eruptions in older people.

◆ The effects of the aging process, especially in the skin, on certain allergic skin diseases is a subject which has received scant attention in the geriatric literature. The skin in older persons undergoes many changes. It becomes drier, thinner, and rougher, has less turgor, and appears flabbier due to gradual loss of elasticity and underlying fat. Circulatory and degenerative diseases also have an adverse effect on the condition of the skin. There is less tendency to acute inflammation, but more of a tendency to scaling and to persistent pruritus. Thus the severe pruritus associated with certain allergic dermatoses tends to persist and to be more resistant to therapy in older than in younger skins.

Occurrence of allergic dermatoses depends on (1) opportunities for exposure to allergenic sensitizing agents and (2) capacity of the individual to undergo those immunologic alterations which express themselves clinically as allergic eruptions. Opportunities for allergenic exposure are likely to be somewhat different in older people. For example, aspirin is used commonly by all age groups, but barbiturates, Butazolidin, rauwolfia, and drugs for hypertension are employed more frequently in older people.

The capacity to undergo allergic sensitization appears to be diminished in older persons, as shown by clinical and experimental evidence. For example, in 1936 Sulzberger and Baer attempted to sensitize a group of elderly patients with picryl chloride with almost completely negative results.[1] This same substance, a powerful sensitizing agent in experimental animals,[1] produced many sensitizations when injected into the skin of a younger group of patients.

Allergic alterations of the skin play an important causal or contributory role in many skin diseases, most of which are not thought of as "allergic dermatoses." In syphilis, tuberculosis, fungous infections of the skin, impetigo, measles, lymphogranuloma venereum, and in numerous other conditions, the disease either could not occur without allergic sensitization to the causal agent or the course of the disease would be quite different. We shall restrict this paper to a discussion of three of the four dermatologic entities which are generally referred to as the allergic dermatoses—allergic eczematous contact dermatitis, allergic urticaria, and allergic drug eruptions. The fourth dermatosis, atopic dermatitis,

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persists rarely after the age of 50 and will not be included here.

Allergic Eczematous Contact Dermatitis

Allergic eczematous contact dermatitis due to both occupational and nonoccupational agents occurs much less frequently in older than in younger persons. The striking reduction in incidence is due to three main factors: (1) relative lack of exposure to certain agents, (2) reduced susceptibility to sensitization, and (3) loss of many allergic contact sensitizations with advancing age.

Allergic contact dermatitis in older people, even during the acute stage, shows relatively little inflammation. Scaling is apt to be especially pronounced in the subacute phase as well as in the chronic phase in which the skin is also thickened, hyperpigmented, and lichenified. The associated itching may become highly disturbing during any phase of the eruption.

Allergic contact dermatitis usually begins and localizes mainly in areas of major exposure. Secondary nonallergic factors such as sweating, heat, friction, and impaired alkali-neutralizing capacity, often assist in causing the eruption and in determining its localizations.

Some of the most common allergens such as plants are much less important in the older age groups than in younger persons. However, dermatitis from certain air-borne allergens, such as ragweed pollen oleoresin, appears to be somewhat more common. The seasonal occurrence of the dermatitis, together with the typical involvement of the "exposed" areas—severe erythema, with lichenification, thickening, moderate edema, and deep furrowing—make the diagnosis relatively easy.

Cosmetics are often used to mask the stigmata of approaching age. Hair dyes are among the more common contact allergens in persons over 50.

Wearing apparel is another common cause, involving those parts which are in immediate contact with the allergenic material, especially where such secondary factors as sweating and friction increase opportunities for exposure. A typical picture may be seen in menopausal women in whom dresses produce involvement of the axillae, cubital spaces, inner aspects of the thighs, and popliteal spaces. Fur-lined or rubber gloves may cause an allergic contact eczema of the hands which may resemble "housewife's eczema" in which irritation from soaps or detergents plays an important part. The dryness of the skin of older persons may become aggravated through excessive or even normal use of toilet soaps, soap powders, and household detergents—a nonspecific type of damage which may predispose them to allergic contact sensitizations.

Topical medicaments, especially local anesthetics and antihistamines, are among the major causes of allergic contact dermatitis. Among the other causes are quinine and resorcin in hair tonics and lotions; mercury, tars, and other ingredients in medications prescribed commonly by physicians and in self-prescribed preparations; pilocarpine given for treatment of glaucoma, producing dermatitis of the eyelids and adjacent parts; nose drops containing such drugs as sulfonamides, penicillin, epinephrine, and antihistamines, producing dermatitis of the upper lip and nostrils; and medications used for pruritus of the anal, vulvar, and scrotal region. Even such generally harmless materials as lanolin and other oils and fats in ointments or creams, which older people use frequently for lubrication of dry and scaly skin, may become causes of allergic dermatitis.

Older people may get contact dermatitis from the materials used in hobbies to which they have turned after they have given up their regular occupations. In persons who spend a great deal of time in gardening, plants, insecticides, fertilizers, etc., should be suspected; stamp col-

lectors may develop an eruption on the fingers due to glue; those who spend their time in clerical work, carpentry, bookbinding or other hobbies, are exposed to the particular materials used in the respective occupations. Painting which has recently become a favorite pastime among older people exposes them to pigments, oils, and turpentine. Contact stomatitis and dermatitis around the mouth may be due to materials used in dentures, cleansing powders for dentures, mouth washes, and tooth pastes.

The tracking down of causal agents requires a certain amount of detective spirit on the part of the physician. In some cases the localization of the eruption alone may tell the story, but most of the time a carefully taken history is absolutely essential. New exposures as well as contact to materials to which exposure has occurred for a long time have to be considered. Patch tests are of great help in finding causal agents, if performed according to standard technics.[2]

TREATMENT

The most important single therapeutic procedure in every case of allergic contact dermatitis is to find the causal agent and then to institute the proper measures for its avoidance and elimination. Specific hyposensitization by the parenteral and oral method is not feasible or practical in the vast majority of cases. Exceptions are cases of chronic or acute recurrent dermatitis due to oleoresins in pollens, plants, and fruits. Specific hyposensitization treatment should never be given during an acute attack of dermatitis, since there is no conclusive evidence that it is effective, while in some cases there is much proof that it aggravates the disease. For hyposensitization injections in sensitization to poison ivy, oak, and sumach, alum precipitated pyridine extracts[3] are recommended. For specific hyposensitization with all other plant oleoresins we recommend the oral method of Shelmire, using oleoresin extracts dissolved in corn oil.

Among nonspecific forms of treatment, suitable topical measures are generally most effective. The principles to be followed are the usual ones for treatment of eczema. In the acute stage it should be very mild: wet compresses with Burow's solution 1:20 in water, boric acid solution, or normal saline solution are often helpful. They may be alternated with bland lotions or emulsions. An example is the following lotion which is preferable to the rather alkaline and thin calamine lotion:

menthol	0.3 gm.
phenol	0.6 gm.
zinc oxide	
talc āā	20.0 gm.
glycerin	10.0 cc.
alcohol	
water āā	35.0 cc.

Any irritating topical medication is to be avoided. Hydrocortisone preparations have proved to be helpful and thus far no case of allergic eczematous sensitization to hydrocortisone itself has been reported. In the acute stage, hydrocortisone, 1 to 2.5 per cent lotions, creams, or ointments are often effective except when there is pronounced vesiculation. In the subacute and chronic stage, these preparations as well as tar-containing medications are indicated. Topical agents containing local anesthetics and antihistamines or other agents with strong eczematogenic sensitizing properties are contraindicated in allergic contact dermatics.

The oral or parenteral administration of antihistamine drugs, however, is often beneficial in reducing the susceptibility to itching and in helping the patient to get some sleep during the night. Aspirin also is effective in some cases in reducing itch susceptibility. In exceptionally severe and widespread cases, oral cortisone or ACTH injections may have to be considered to help the patient over the disabling stages. Such treatment, of course, is suppressive and not curative.

Drug Eruptions

A drug eruption is any eruption which is produced by a drug no matter what the route of administration. The drug reaches the skin in most cases from within via the blood stream after oral administration, parenteral injection, injection into sinuses or body cavities for diagnostic or therapeutic purposes, or inhalation. It has been pointed out that topical medicaments are among the most common causes of allergic contact dermatitis. Such cases, of course, can be classified both as drug eruptions and as allergic contact dermatitis. Absorption of medications from mucous membranes or from the skin after topical application may also cause drug eruptions which are identical in appearance with those elicited by systemic administration. Recently, even a "fixed" drug eruption was described due to absorption of dioxyanthranol after topical application.[4]

Drug eruptions are seen rather frequently in people over 50, perhaps because many of them require medications or because they have acquired the habit of taking drugs. The over-the-counter sale of patent medicines and their exaggerated and continuous advertising are probably responsible for many drug eruptions. In searching for the cause of an eruption, any "new" drugs or new combinations of drugs should be suspected; "old" drugs, however, taken regularly for months or years, must also be considered suspect. The reasons why allergic sensitization to drugs may take place suddenly after years of noninjurious exposure are still unknown.

Drug eruptions are varied in their clinical appearance and can mimic almost any known variety of skin disease. Eruptions may vary from small lesions, such as a postarsenical epithelioma, to those covering the integument, such as an exfoliative erythroderma from a barbiturate.

Not all drug eruptions are based on an allergic mechanism[5] but many of those seen in general practice are of the allergic type. Table 1 presents some of the important differences between allergic and nonallergic drug eruptions.

The clinical picture is usually not sufficient to permit the physician to specify the particular drug which has caused the eruption, since many drugs may produce the identical clinical picture. Moreover, a single drug can elicit many varieties of eruptions in different individuals. Occasionally, a single drug may engender more than one variety in the same person on different or even the same occasions. When the drug is carried in the bloodstream, the eruption tends to a wide and symmetrical distribution. In a small minority of cases, the cutaneous changes are accompanied by fever, headache, malaise, nausea, and joint symptoms. Allergic drug sensitivities also may cause leukopenia, agranulocytosis, aplastic anemia, and thrombocytopenic purpura.

The most important subjective symptom of drug eruptions, and often the

TABLE 1

SOME DIFFERENCES BETWEEN NONALLERGIC AND ALLERGIC DRUG ERUPTIONS

Point of difference	*Nonallergic*	*Allergic*
Clinical appearance	quite uniform	many different types of eruption
Quantity necessary to produce eruption	relatively large	usually small or minute
Cumulative effect	often necessary	usually not necessary
Association between cutaneous effects and characteristic pharmacologic action	often	unrelated
Same cutaneous effect produced by pharmacologically different drugs	no	very often
Incidence of reactions among those exposed	occurs in many or most or all	usually occurs in relatively few

only evidence of drug intolerance, is pruritus but many such eruptions have no subjective symptoms. In older patients complaining of unexplained itching without an eruption, one should be alert for a possible drug causation, in addition to pruritus senilis and symptomatic pruritus as in diabetes, liver or kidney disease, blood dyscrasias, lymphomas, and internal malignancies.

Almost any drug may cause allergic drug eruptions, but there are certain drugs which have a higher sensitizing capacity than others. The knowledge of this fact should make the physician alert to the sensitizing capacities of the many drugs which a patient may have taken. Some drugs have a propensity for causing certain types of eruptions and certain types of eruptions are more likely to be caused by certain drugs, as shown in tables 2 and 3. These listings are by no means complete, however, and it is not known yet in certain forms of drug eruptions whether they are based on an allergic sensitization mechanism.

The so-called "fixed drug eruption" is one type which is not sufficiently recognized among nondermatologists. It presents single or multiple, round or oval, sharply demarcated, erythematous to bluish patches, which, as they undergo involution, gradually become darker and finally leave brownish hyperpigmentation. In rare instances these patches are bullous, vesicular, or urticarial. Upon readministration of the offending drug, the same sites become reactivated, developing an acute erythematous, slightly edematous border around the hyperpigmented center. New "fixed" lesions may appear simultaneously with the flare-up of the original and older sites.

A painstaking history and a high index of suspicion are vital in locating the causes of drug eruptions. Rather than ask "Do you take any drugs?" one should be very specific in questions to the patient. Questions which are more likely to elicit the desired information are:

TABLE 2*

SOME COMMON ERUPTIONS AND EXAMPLES OF THEIR CAUSAL DRUGS

Acneform and furunculoid eruptions: androgenic hormones, bromides, iodides, etc.

Eczematous eruptions: antihistamines, ephedrine, formalin, Furacin, local anesthetics, mercurials, penicillin, quinine, etc.

Eruptions resembling erythema multiforme: antipyrine, Aureomycin, barbiturates, penicillin, phenolphthalein, salicylates, sulfonamides, etc.

Eruptions resembling erythema nodosum: bromides, iodides, salicylates, sulfonamides, etc.

Fixed eruptions: antipyrine, aspirin, Atabrine, barbiturates, gold, para-aminosalicylic acid, Phenacetin, phenolphthalein, sulfonamides, etc.

Lichenoid eruptions and eruptions resembling lichen planus: amphetamine sulfate, arsenicals, Atabrine, gold, etc.

Pemphigoid eruptions: arsenicals, bromides, iodides, mercurials, etc.

Vegetating and tuberous eruptions: bromides and iodides.

Purpuric eruptions: aspirin, barbiturates, Butazolidin, carbamides, Chloromycetin, iodides, p-aminosalicylic acid, sulfonamides, etc.

Exfoliative dermatitis and eruptions resembling exanthems: arsenicals, barbiturates, belladonna, digitalis, mercurials, penicillin, quinidine, salicylates, streptomycin, sulfonamides, etc.

Urticaria and angioneurotic edema: aspirin and other salicylates, atropine, barbiturates, belladonna, bromides, insulin, iodides, liver extract, morphine, and other opiates, penicillin, phenolphthalein, etc.

*Based on M. B. Sulzberger and R. L. Baer: Office Immunology, Year Book Publishers, Chicago, Ill., 1947.

"What do you take to help you sleep?" "Which laxatives and physics?" "What headache medications?" "Cold tablets? aspirin? nerve medicines? effervescent drinks? tonics, and so on?" "Have you consulted a physician or dentist recently? What did he prescribe for you or use on you?" "When was the last time you took any medication?"

A history that a certain medicament has been taken for a long time or at long intervals does not rule out that it finally has caused an allergic reaction. One should inquire about medicated chewing

TABLE 3*

COMMON DRUGS AND SOME EXAMPLES OF ERUPTIONS THEY PRODUCE

Atabrine
- Alopecias
- Eczematous and eczematoid
- Exfoliating erythrodermas
- Fixed
- Resembling lichen planus
- Resembling lichen pilaris
- Pigmented and depigmented
- Resembling poikiloderma
- Ungual dystrophies

Barbiturates
- Eczematous and eczematoid
- Resembling erythema multiform
- Erythrodermas
- Resembling exanthems
- Exfoliative dermatitis
- Fixed
- Gingival lesions
- Mucous membrane lesions
- Pemphigoid

Belladonna
- Resembling exanthems
- Purpuric
- Urticarial

Bromine and iodine
- Acneform
- Angioneurotic edemas
- Resembling ecthyma
- Eczematous and eczematoid
- Resembling erythema multiforme
- Resembling erythema nodosum
- Resembling exanthems
- Furunculoid
- Pemphigoid
- Purpuric and hemorrhagic
- Tuberous and fungating
- Ulcerative
- Urticarial

Butazolidin
- Resembling exanthems
- Purpuric
- Urticarial

Digitalis
- Erythematous
- Resembling exanthems
- Papular

Opium derivatives
- Resembling exanthems
- Pruritus
- Urticarial

Penicillin
- Angioneurotic edema
- Eczematous and eczematoid
- Resembling exanthems
- "Id," pompholyx and dyshidrosis-like eruptions of hands, feet
- Resembling serum sickness with joint swellings and fever
- Resembling tinea cruris
- Urticarial

Phenolphthalein
- Eczematous and eczematoid
- Resembling erythema multiforme
- Fixed
- Pemphigoid
- Urticarial

Quinidine
- Eczematous and eczematoid
- Photosensitization eruptions in exposed areas

Salicylates
- Angioneurotic edema
- Conjunctivitis
- Eczematous and eczematoid
- Erythemas
- Resembling erythema multiforme
- Resembling erythema nodosum
- Fixed
- Resembling exanthems
- Pemphigoid
- Resembling pompholyx and dyshidrosis
- Urticarial

*Based on M. B. Sulzberger and R. L. Baer: Office Immunology, Year Book Publishers, Chicago, Ill., 1947.

gum, candies, and other items, such as hangover medications, and vitamins.

It is worth remembering that barbiturates are contained not only in "sleeping pills" but also in many patent and "ethical" drugs, including drugs for hypertension, reducing pills, antiasthmatic tablets, and remedies against nervous exhaustion and heart conditions.

In short, good detective work, although usually time consuming, is often necessary to obtain a complete drug history. The reward, however, is well worth the effort when the patient is finally relieved from distressing and sometimes incapacitating eruptions.

Skin tests are generally of no value in establishing etiology except in eruptions that are eczematous in character and in a small minority of urticarial eruptions. In

patients with eczematous eruptions due to such drugs as mercury, quinine, and sulfonamides, a positive patch test usually confirms the culpability.

Tentative proof is obtained when continued avoidance of a drug is followed by definite improvement within a few days to several months. Final proof can be obtained only when the readministration of the suspected drug causes a recurrence. It is obvious that in order to avoid unnecessary suffering or dangerous reactions, such readministration should be carried out only if it is essential to have this proof. In certain manifestations of a serious character, such as anaphylactoid reactions, exfoliative dermatitis, and laryngeal edema, administration of the drug is, of course, strictly contraindicated.

TREATMENT

Elimination and avoidance of the causal drug will be followed by clearing of the eruption in most cases. If the lesions and accompanying symptoms are acute they are likely to disappear within a few days. However, certain drug eruptions, especially those associated with chronic skin changes, may linger on for months despite withdrawal of the drug. Among these are eruptions due to arsenic, bromides, iodides, and sulfonamides and the often seen urticarial eruptions from penicillin. Occasionally, as in cases of allergic insulin hypersensitivity, it may not be possible to withhold the drug, since the patient must have it for other medical reasons. In such cases one should try to change to a different type of insulin, such as protamine zinc insulin, or to insulin prepared from a different animal species, or one should attempt hyposensitization. This is worth while only in exceptional instances and if there is previous evidence that successful hyposensitization has been achieved with the particular drug. In most cases it is possible to terminate use of the offending drug and to substitute a drug of different chemical structure. For example, in eruptions due to barbiturates, chloral hydrate, bromides, antihistamines, codeine, Demerol, and so on, may be substituted. Cascara, saline laxatives, senna, and milk of magnesia may be substituted in allergic eruptions due to a laxative containing phenolphthalein.

Specific hyposensitization is generally not practicable in treatment of drug eruptions. Among the exceptions where parenteral or oral hyposensitization have been recommended are some cases of cutaneous allergy to insulin, penicillin, liver extract, and sulfonamides.

A potentially important form of nonspecific treatment is the administration of substances which hasten the excretion of the offending drugs. An outstanding example is the intramuscular administration of dithiopropanol (BAL) in cases of allergic reactions due to certain metals such as arsenic, mercury, bismuth, antimony, and chromium. Much less striking is the effect of sodium chloride in eruptions due to iodides and bromides.

Symptomatic topical and systemic treatment must be prescribed in every case of drug eruption which is associated with pruritus or with lesions which are susceptible to such measures. Symptomatic treatment in drug eruptions not only affords the patient relief, but shortens the period of disability and disfigurement and in some cases is actually lifesaving. All known topical measures, such as baths, lotions, and ointments, should be prescribed on the basis of the general principles of symptomatic dermatologic therapy. The systemic administration of such drugs as salicylates and antihistamines is helpful against pruritus but the physician must be aware that these drugs themselves may cause superimposed drug eruptions. Another complicating phenomenon is allergic group-hypersensitivity or cross-sensitization. Here the patient has become allergic not only to the drug to which he has been exposed but also to one or more chem-

ically related drugs.[6] In order to avoid reactions due to cross-sensitization, drugs prescribed for symptomatic relief should preferably not be chemical relatives of the suspected medication.

Administration of ACTH or steroid hormones is warranted in incapacitating drug eruptions only if the particular type of lesion is known to be susceptible to this form of therapy. Barring the usual medical contraindications, these eruptions form an ideal indication for steroid therapy because they are likely to be self-limited and the hormone therapy can be gradually discontinued after a period of days to months.

Urticaria

Diagnosis of urticaria can be made only if spontaneously appearing wheals and large angioneurotic swellings are the only types of lesions seen. If wheals are part of an eruption together with other cutaneous lesions, other diagnoses must be considered in which urticarial lesions are only part of the clinical picture. Among these are insect bites, urticarial dermographism, dermatitis herpetiformis, multiform erythemas, and others. In true urticaria, the lesions, except for large angioneurotic swellings, develop and regress quickly in four to six hours. Lesions which persist for twenty-four hours or longer are not part of true urticaria.

It is important to differentiate between acute and chronic urticaria, although an absolute delimitation is, of course, not feasible. Acute urticaria usually includes an eruption which lasts from a few hours to three weeks. This type of urticaria tends to be less common in persons over 50, perhaps because most people of this age have discovered those foods, commonly used drugs, and other substances which tend to cause acute urticaria. Particularly in acute urticaria the layman often does not find it difficult to discover the eliciting agent because of the obvious time relationship between exposure to the allergen and the sudden onset of the urticarial attack. Another reason for less frequent occurrence after 50 is a less desire toward undergoing sensitization.

Chronic urticaria is a major dermatologic problem in persons over 50. Once an urticarial eruption has continued for three or more months it is impossible to foretell how much longer it will last. Even in some cases where the causal agent is known and is avoided, as in penicillin urticaria, the eruption has been seen to persist for twelve, eighteen, and more months. The reasons for such a prolonged course, in the absence of re-exposures to the causal allergen are not well understood. It is possible that cross-sensitization to secondary allergens, immunochemically related but unknown, plays a role in such persistent cases.[6]

In our patient material we have been able to discover specific causes in only a small minority of cases of chronic urticaria. Other authors have been much more fortunate or much more ingenious in ferreting out the causes. The three major groups of causal factors are:

1. *Drugs.* This group is the single most common cause in our material. The most common culprits among the medications are listed in table 1.

2. *Foods.* Older people usually have learned from experience which foods to avoid. New allergic sensitizations to one or more foods, however, may develop at any time. The most common allergenic foods are said to be eggs, fish, shellfish, wheat, nuts, chocolate, and tomatoes. Food urticaria must be differentiated from eruptions consisting of urticarial lesions combined with erythema multiforme-like eruptions, accompanied by fever and other general symptoms which are produced occasionally by spoiled foods or the micro-organisms therein.

3. *Foci of infection.* Every patient with chronic urticaria should be carefully examined for possible infections in the sinuses, teeth, tonsils, prostate or female genital tract, gallbladder, or elsewhere. It is not known whether foci or infection are causal or merely help to maintain urticarial eruptions.

There are many other etiologic groups which are known to have produced chronic urticaria. Among these, intestinal

parasites are perhaps the most frequently overlooked. Cases of chronic urticaria have been reported in which intestinal parasites were demonstrated and in which eruption ceased subsequent to treatment with vermifuges.[7] Among the other less common causes are inhalant allergens,[8] physical agents,[9] and autochthonous substances such as hormones[10] and acetylcholine. In our experience, emotional factors are not among the causes of urticaria but, along with a multitude of other equally important secondary factors such as friction, temperature changes, and pressure, may in some cases become contributory. As we have said, a specific cause cannot be found in the large majority of cases.

In our experience, skin tests for the immediate wheal response by the intracutaneous or scratch method fail to reveal causal factors in most cases and one must rely on other measures in the search for allergenic agents. Again a painstaking history is of utmost importance. If exacerbations of the eruption coincide with exposure to a certain drug, food, or inhalant, and remissions coincide with avoidance of the same agent, such a possible causal agent must be strictly avoided. Even if no immediate benefit is apparent, abstention should be extended for several months, since urticarial eruptions may continue long after avoidance of the causal substance. Usually, however, there will be significant improvement after several days to weeks.

When a careful physical examination reveals a possible focus of infection, treatment should be directed toward suppression—using antibiotics, but not penicillin—or cure of the infection. In many cases, however, the surgical measures necessary to cure a focus are out of proportion to the chances of a beneficial effect on the urticaria. Here the physician must make his own decision.

The only notable recent addition to the therapeutic armamentarium against urticaria are the corticosteroids and ACTH. While large doses of these agents are helpful in some patients, they have not lived up to the original expectations and they certainly are not the answer to the nonspecific management of urticaria. Moreover, particularly in the older age groups, the possibility of serious side effects cannot be overlooked. In general, therefore, the physician has to rely on other measures. The single most outstanding group of drugs for prolonged relief in urticaria are the antihistamines. Their subcutaneous and intramuscular use should be considered in cases in which oral administration has proved ineffective. For immediate temporary relief epinephrine 1:1,000 solution can be given but the possible beneficial effects must be weighed against undesirable reactions. Antipruritic topical medicaments, ephedrine sulfate, and mild laxatives which do not contain phenolphthalein or bromides are among other measures which can be tried.

The pyridine extracts mentioned on page 267 are available from Strong Cobb Company, Inc., Cleveland, Ohio. The oleoresin extracts dissolved in corn oil (page 267) are available from the Graham Laboratories, Dallas, Texas.

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The physiology of the aging process in the musculoskeletal apparatus

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This study brings into focus certain aspects of the physiology of the aging process of the musculoskeletal system which are sufficiently integrated to apply to the problems of geriatric orthopedics. It is especially concerned with the underlying physiologic processes which condition the musculoskeletal disabilities of the aged. Progress in this field will follow development of possible preventative measures far better than it will attempts to reconstruct the structural damage which has already occurred.

◆ Except for certain disease processes more frequent in later life, most of the musculoskeletal disabilities of the aged are tissue responses to metabolic or endocrine changes of a systemic nature. These can be measured, isolated, and may some day be controlled. Although systemic in operation, their effect on the major musculoskeletal tissues is rather specific. The local structural disability which follows is best viewed as the combined failure of these weakened tissues to withstand the normal stresses and strains for which the musculoskeletal apparatus is intended. To this structural weakness each of the major tissues of the apparatus contributes its own share. They must therefore be studied separately before their failure is understood collectively.

Cartilage

The cardinal point in any consideration of the physiology of articular cartilage is its histologic avascularity, an observation classically recorded by Toynbee in 1841.[1] Cartilage is composed chiefly of a homogeneous fibrillar substance produced early in life by or around the cartilage cell. This cell is sparsely distributed through articular cartilage in its adult state. As a tissue, its function depends upon the chemical integrity of its intercellular substance, a glycoprotein related to the collagen of bone and connective tissue with which chondroitin sulfuric acid is uniquely combined.[2,3]

The internal avascularity obviously signifies a low nutritional requirement, especially since no other portals of entry for replacement material have been convincingly demonstrated.[4] This further suggests a relatively fixed tissue, chemically stable, kept viable by a low order of cellular activity. In the adult state, active cartilage cells are found almost exclusively concentrated at the chondro-osseous border—the residual basal cells of the growth period. Unless these cells are destroyed by trauma or disease, the tissue can outlast by many years the far more labile bone. The metabolic stability of cartilage bespeaks its great phylogenetic age, as does its early embryonic development.

The persistence of large surfaces of articular cartilage in the aged is a phe-

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nomenon not often noted. Degeneration of fibrocartilage within the intervertebral spaces is almost uniform in later life.[5] However, this is not hyaline cartilage; its histology and its physiology are of a different order. The hyaline covering of the vertebral surfaces remains remarkably intact.[6] Small isolated areas of necrosis found in traumatized joints are common enough, and similar areas beneath the inflamed pannus of arthritic joints are well recognized. These and similar examples of localized chondromalacia due to specific trauma or disease do not support a concept of generalized cartilage degeneration progressing with age. Observations of elderly joint surfaces prove quite the contrary.

During the past decade a large number of femoral heads have been observed or removed from elderly patients during the course of arthroplastic procedures or prosthetic replacement for ununited fractures of the hip joint. Often in such specimens, in spite of serious avascular necrosis of the adjacent subchondral bone, the articular cartilage appears well preserved over most of its surface. Similarly, during the course of debridement of the elderly knee joint disabled by osteophytes and massive hypertrophic synovitis as suggested by Magnusson,[7] the same observation may be made of the articular cartilage of the femoral and tibial condyles.

The chondromalacia seen in inflammatory or posttraumatic states is apt to be confined to one or more small areas and is apparently a response to specific injury or to the wear and tear of abnormal joint mechanics.[4] They are therefore found far more frequently in the joints of the lower extremities than in the nonweightbearing upper extremities. These localized, usually quiescent, necrotic areas in the articular surface persisting after the immediate symptoms of trauma or inflammation have subsided are not in themselves disabling lesions in the elderly. Certainly they do not indicate a general physiologic disruption of the tissue as part of the aging process. Most of the nontraumatic joint disturbances of the elderly will be found related to disruption of the subchondral bone substance rather than to attrition of its hyaline cartilage covering.

In view of the many contradictory reports, it is necessary to emphasize that this study deals with the process of physiologic senescence. It does not refer to the relative frequency of specific trauma or inflammatory joint disease in the elderly patient whose life has been exceptionally rugged, or that in the obese patient suffering from mechanically abnormal wear and tear of the articular tissues. In the physiology of the normal aging, musculoskeletal apparatus articular cartilage is found to be the most stable and least contributory to disability and dysfunction.

Bone

Normal osteogenesis and reparative osteogenesis take place by means of two processes acting more or less concurrently. The first of the processes is ossification —that is, formation of bone trabeculae— and it depends biochemically on the synthesis of a complex collagen-type bone protein. This is a preconditioned tissue formation replacing the primitive cartilage formed within the mesenchymal anlage of the embryonic skeleton. The detailed histology of this process is well described in the definitive publications of Urist and McLean.[8] Of the biologic forces concerned in the making of bone tissue, we know nothing, however much we know of its chemistry and its developing forms. The importance of this process to the human economy is demonstrated in the fact that 20 per cent of the body protein is used in the making of bone matrix.

When this protein trabecular framework is laid down, the nature of its chemical composition is such as to precipitate immediately an equally complex

inorganic bone salt composed of minerals otherwise soluble in the blood stream. This precipitation, the second of the parallel processes, is generally referred to as calcification. The actual process of histologic ossification without calcification was demonstrated experimentally by Ham, Tisdall, and Drake in rachitic rabbits.[9] In similar fashion, reparative osteogenesis is primarily a histologic process of ossification depending upon a continuing synthesis of bone protein followed by precipitation of bone salt to the extent to which the mineral elements are available in the blood stream.

That bone exists as a labile tissue undergoing continuous simultaneous deposition of new molecules and absorption of the old is an observation first introduced by John Hunter in the eighteenth century and confirmed repeatedly by succeeding osteologists. This constant replacement and absorption of bone elements supported by an extensive intraosseous vascular apparatus must be fully comprehended in any consideration of bone physiology or pathology. Normal bone demands a balance between these two biologic processes. In the presence of adequate chemical synthesis, these processes are inexorable. Any metabolic or endocrine disturbance of production or any dysfunction in the vascular distribution of its chemical elements must influence bone structure by interfering with this balance.

The fundamental characteristic of the normal aging skeleton is not, as x-ray evidence would suggest, the lack of mineral salts, but is an imbalance in the deposition-resorption equation of trabecular molecules. In the presence of a normal diet, the existing trabeculae of aging bone are adequately calcified; it is the decrease in the density of trabeculae per volume that forms the characteristic picture of senile osteoporosis.[10]

In the growing child, rate of deposition of bone exceeds that of resorption. During early adult life, the two processes are generally in balance in the normal subject. In the aged, trabecular absorption takes place normally but the synthesis and deposition of bone protein diminishes.[11,12] In a more generic sense in relation to the physiology of bone, anabolic activity diminishes toward the later years and catabolic activity either remains normal or perhaps may be even accelerated. The effect of this negative osteogenic balance is that less bone substance is present and there is less matrix to combine with bone salt. It is the combined bone-salt-proteinate which gives rigidity and strength to the skeleton.

Endocrine and metabolic deficiencies and systemic decrease in the peripheral vascular supply are the physiologic substrata of diminishing bone production in the aging skeleton. These deficiencies and dysfunctions interfere with the synthesis and distribution of the chemical elements needed for the production of bone substance. Clinically speaking, because of these deficiencies aged bone is soft and it is brittle. These two physical defects condition most of the commonly manifested problems of the aging skeleton. Among them are generalized osteoporosis, avascular necrosis at areas of maximum stress, fractures at the brittle neck of the femur, compression of the vertebral body, and, in the presence of faulty joint mechanics, collapse of subchondral bone.[13]

Muscle

The characteristic of an aging muscular apparatus is atrophy of disuse. Histologic changes in the muscle cell and its interstitial framework have been sought and so far have not been of sufficient order to be significant. Muscle tissue removed at the operating table from elderly patients appears quite normal under the microscope. Diminished strength lies in the failure of the neural control mechanism, in the lessened rapidity of chemical reactions involved in muscle contrac-

tion, and in the decreased ability of the vascular tree to deliver quickly blood stream elements needed to replace those used up in muscle activity. These lead to atrophy of disuse.

Muscle tissue, unlike bone, is a histologically fixed quantity; unlike cartilage, it is neither chemically nor metabolically stable.[14] Its decreased activity in the aged is incidental to decreased metabolic, neurogenic, and vascular factors. As it is, the remarkable relative strength of muscle contraction among old men otherwise healthy and under sufficient incentive has been observed often enough outside the laboratory.[15] Among the problems of geriatric orthopedics, the muscular system is of little concern. Its gradual failure, clinically speaking, is most often a failure of the circulatory mechanism or central nervous system.

Conclusions

The chief physiologic problem of orthopedic geriatrics is the synthesis of bone protein. Articular cartilage is a relatively avascular tissue which, when not injured by major trauma or the repeated minor traumata of mechanically unsound joint function, is apt to persist undamaged well into the late decades. Muscle tissue remains all but unchanged except as its power is influenced by circulatory or central nervous system deterioration.

The skeletal deformities of the aged are far more often due to the collapse of soft and brittle bone at its points of major stresses. When failing osteogenesis in the aging human can be controlled or retarded, his orthopedic defects will be largely prevented. In its physiologic aspect, this is chiefly a problem in protein synthesis within the senescent skeleton.

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Neuromuscular diseases of later maturity. Part II

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◆ Among the many neuromuscular diseases which have their onset in later maturity is a wasting and weakness of muscle which occurs in association with toxic activity of the thyroid gland.

Chronic Thyrotoxic Myopathy

The realization that wasting and weakness of striated musculature may accompany thyrotoxicosis has been evident in the medical literature only in recent years.[43] For some time, myasthenia gravis and thyrotoxicosis have been known to occur in the same patient and a causal relationship had been assumed;[44] however, recent studies indicate that they are separate disease entities.[43] Periodic paralysis and thyrotoxicosis may sometimes occur together, and there appears to be a causal relationship since the paralysis seems to disappear when the euthyroid state is reached.[45] Ophthalmoplegia and exophthalmos with hyperthyroidism are well known but are not fully understood.[46] Hypothyroidism may result in changes in the contractile mechanism of muscle, resembling that encountered in myotonia.[47] Thus, thyroid dysfunctions seem to be able to produce various neuromuscular disturbances, with an effect primarily upon intrinsic muscle metabolism.[45]

Millikan and Haines[43] described 9 cases of chronic thyrotoxic myopathy, of which 4 began after the age of 50. One case occurred in a man of 66. Thorn and Eder[48] noted a tendency for the condition to develop in older age groups. Millikan and Haines[43] found certain findings significant in such cases. Distribution of muscle wasting and weakness was most common in the pelvic girdle, although the shoulder girdle and limb muscles could also be involved. The deep reflexes of involved muscles persisted or were even hyperactive. Neostigmine and curare tests were negative. The electromyogram was reported as being normal in 3 cases. They felt that the syndrome of "acute thyrotoxic myopathy" was too poorly substantiated in the literature to be considered as an entity, and emphasized the *gradual* involvement of the muscles. They pointed out the necessity of careful establishment of the diagnosis of thyrotoxicosis by such means as metabolic rate determination, radioactive iodine tracer studies, and protein-bound blood iodine determinations. The final proof is recovery of damaged muscle function following appropriate therapy for the thyrotoxicosis, as this occurs in no other neuromuscular condition. When myasthenia gravis and thyrotoxicosis occur in the same patient, control of the thyroid disease first will facilitate care of the myasthenia gravis.

Case 6. F. M., a 68-year-old white male, was admitted to the medical service of the University of Pennsylvania Hospital on October 20, 1953, at the request of Dr. Edward Rose. He had had pain in both shoulders in July 1952, which was attributed to a bursitis and which responded slowly to various forms of treatment. Over the

preceding eight months, he had noted gradual weakness in the muscles of the upper extremities. His weight had dropped from 108 lb. despite an increased food intake. He denied heat intolerance, but did complain of a fine tremor of his hands, occasional episodes of profuse perspiration, and infrequent palpitations.

General examination showed a pulse rate of 110 per minute and a blood pressure of 140/60, with other vital signs well within normal limits. The thyroid gland was felt to be diffusely enlarged to about twice its normal size. Neurologic examination revealed generalized muscular wasting with the calf muscles least involved. Power impairment was slight in arms and forearms, while the lower extremity muscles appeared in good condition. He could step up onto a chair without difficulty, and walked well on toes and on heels. Fasciculations were observed in the vastus medialis and gastrocnemii. Deep and periosteal reflexes were present and equally active. There were no pathologic reflexes. No sensory disturbances and no change in the functions of the cranial nerves were noted.

Blood count was within normal limits. Occasionally the urine showed a 2 to 3 plus sugar. Blood Kolmer and Kline tests were nonreactive, blood urea nitrogen was 12 mg. per cent, and fasting blood sugar, 116 mg. per cent. Total serum cholesterol was reported as 184 mg. per cent and 155 mg. per cent. Basal metabolism was plus 76 per cent. The protein-bound blood iodine was 9.2 gamma per 100 cc. Uptake of I^{131} at the end of twenty-four hours was 69 per cent. Blood serum creatine was 1.66 mg. per cent and serum creatinine, 0.6 mg. per cent. Urine creatine was 0.057 gm. and urine creatinine, 0.629 gm. per twenty-four hours. Glucose tolerance was: fasting, 92 mg. per cent; one-half hour, 184 mg. per cent; and two hours, 300 mg. per cent. An electrocardiogram showed changes suggesting a lesion in the posterior wall. Chest and shoulder roentgenograms were normal.

The neostigmine test – 1.5 mg. neostigmine methylsulphate and 0.4 mg. atropine injected subcutaneously—did not improve muscle power and was felt to rule out the association of myasthenia gravis.

William J. Erdman, M.D., found gross fasciculations in the vastus medialis and gastrocnemii. Chronaxie values of these muscles was within normal limits. Electromyographic recordings of the vastus medialis revealed pronounced fibrillation and fasciculation voltages, normal motor unit potentials, no base line disturbance.

The patient was given a therapeutic dose of I^{131}. A few days later a cardiac fibrillation and ankle edema developed, but these yielded readily to sedation, bed rest, low salt diet, and mercurial diuretics. He was sent home on a high protein, high carbohydrate, and high caloric diet with supplementary vitamins, and according to recent information, he has gained weight, become stronger, and returned to work.

The diagnosis of chronic thyrotoxic myopathy rests essentially upon the physician's awareness that such a relationship can occur. Establishing the diagnosis is important because the muscular effects can apparently be reversed when the thyroid state is adjusted.

Idiopathic Dermatomyositis and Polymyositis

Wasting and weakness of voluntary muscles may result from inflammatory reactions in the muscles themselves. Adams, Denny-Brown, and Pearson[49] described an acute form which is more common in children and a chronic form which is more evident after middle life. Despite the many unknown factors in the late form of myositis and the rarity of the disease, its clinical features are significant if one is to differentiate neuromuscular diseases beginning late in life.

Characteristically, the process manifests itself as an increased fatigability and weakness of the muscles of the lower extremities. Tightness and tenderness of the thigh muscles appear. Leg and foot muscles are said to weaken, then the power in the hands and forearms is lost, and late in the disease, muscles of the neck and cranial nerves may grow progressively weaker. Rarely are there any sensory disturbances. Reflexes are said to be present until late in the disease process. There may be an impairment in the electrical excitability of the involved muscles, but the electrical response to faradic current persists as long as any intact muscle fibers remain. Often the process seems to be a slowly progressive symmetrical weakness, tenderness, and induration of muscles leading to fibrous contractures. The skin at times may show an erythema, induration, and hyperpigmentation which may resemble scleroderma.

Case 7. S. P. H., a white male aged 52, was seen first on June 28, 1940, in the Neurology Outpatient Department of the University of Pennsylvania Hospital. On May 30, 1939, while marching in a Memorial Day parade, the patient first noted ready fatigability and weakness in the lower extremities. He also became aware of a shortness of breath on exercise. His head tended to fall forward, until he nearly had to hold it up with his hands. By September, he felt that the "cords of his neck" were enlarged since he was unable to button his collar and the neck muscles seemed stiff. Injections of Novocain into neck muscles appeared to "loosen up the neck," and the "cords became smaller." Although the difficulty in holding up his head increased, the weakness and fatigability of the lower extremities and the dyspnea on exertion no longer troubled him. The hands showed some weakness and fingers felt cold. He lost his ability to whistle.

The most striking feature on clinical examination was the tendency for the patient's head to fall forward. Blood pressure was 120/90. Heart auscultation showed extrasystoles. There was a slight inguinal adenopathy. Muscles of neck and shoulder girdles showed wasting and weakness, but muscles of trunk and lower extremities did not appear to be weak or atrophied. Arm muscles were of good volume, but those of forearms and hands were atrophic. There were no fasciculations. The patient showed no disturbance in either equilibratory or nonequilibratory coordination, and his sensory system was intact. The cranial nerves were intact.

The skin over the shoulders and arms seemed tough and leathery, but the skin of the hands and, to a lesser extent, of the feet, was cold, moist, and so thick and stiff that finger movements were difficult. Nail beds were cyanotic.

Blood count showed a hemoglobin of 87 per cent; 5,000,000 erythrocytes per cu. mm.; and 13,200 leukocytes per cu. mm., of which 66 per cent were polymorphonuclears, 26 per cent lymphocytes, 5 per cent monocytes, and 3 per cent eosinophils. Blood urea nitrogen was 8 mg. per cent; fasting blood sugar, 89 mg. per cent; total serum cholesterol, 246 mg. per cent; blood serum calcium, 10.6 mg. per cent; and phosphorus, 4.9 mg. per cent. Urine showed a few fine granular casts and many leukocytes. Kolmer and Kahn tests were nonreactive. Basal metabolism was +1 per cent and −12 per cent.

Roentgenograms of hands, skull, cervical and thoracic vertebrae, and chest disclosed no significant abnormality. An electrocardiogram was interpreted as showing auricular fibrillation.

In December 1940, a complaint of cold, painful fingers and hands led to study in the peripheral vascular clinic of University of Pennsylvania Hospital. The right ulnar and left dorsalis pedis pulses were not palpable. An ischemia was demonstrated in fingers and hands, but less in toes and feet, allowing a diagnosis of Raynaud's syndrome to be made.

The patient was fitted with a neck brace for head support and followed in the Neurologic Outpatient Department. He was treated with tocopherols, wheat germ oil, nicotinic acid, and desoxycorticosterone-acetate. He continued to lose weight and became progressively weaker. The wasting and weakness were always more noticeable in the muscles of neck, shoulder girdle, and face, but muscles of hands and upper extremities, of the back, and of the lower extremities were all somewhat involved. The patient expired on June 12, 1942.

Gross findings at autopsy, performed about five hours after death, included bilateral chronic apical fibrous pleurisy; chronic fibrous pericarditis; coronary sclerosis with slight narrowings; old myocardial infarctions of base of posterior left ventricle and right ventricle; focal scarring of lateral and anterior walls of left ventricle; marked dilatation of right auricle and ventricle; bilateral, calcified, apical chronic fibrous tuberculosis; passive congestion of the liver and spleen; hypertrophy of right kidney; congenital hypoplasia with fibrous obliteration of pelvis and ureter of left kidney; and cystitis.

Histologic study showed an old myocardial infarction, pulmonary emphysema, passive congestion of liver and spleen, congenital hypoplasia or healed pyelonephritis of left kidney with fibrous obliteration of left ureter, hypertrophy and pyelitis of right kidney, and chronic cystitis.

In one section of the skin of the hands, the stratum corneum varied in thickness, quite thick in some portions and quite thin in others. The stratum lucidum was never well marked. The stratum granulosum was readily identifiable and varied in thickness from 1 to 5 cells deep. The stratum Malpighii showed nothing unusual. In the papillary layer of the derma, several focal collections of infiltrating cells were seen, composed of plasma cells, lymphocytes, and monocytes (figure 3A). A similar collection was seen deep in the reticular layer of the derma, as well as some striated muscle fibers, a few of which were swollen and hyalinized.

In another section, the epidermal layers were thinned, but the remarkable changes were in the derma. Both papillary and reticular layers were markedly hyalinized (figure 3B). The bundles of collagenous fibers were dense and swollen, with a paucity of nuclei. Sebaceous glands, sweat glands, and hair follicles were intact in this dense, hyalinized derma. No collections of infiltrating cells were noted.

In a third section, the stratum corneum was especially hornified, but the other layers of the

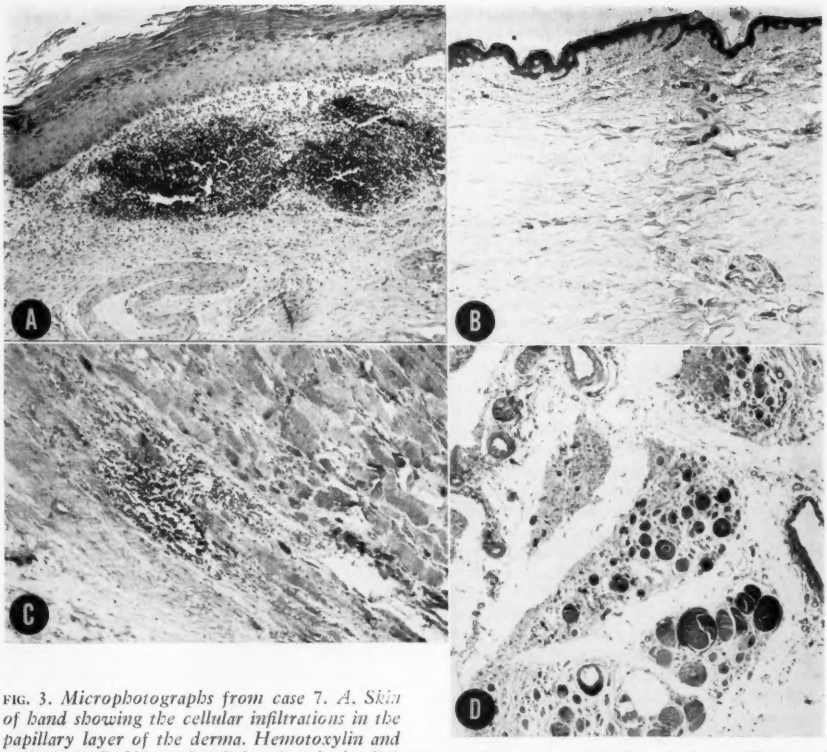

FIG. 3. *Microphotographs from case 7. A. Skin of hand showing the cellular infiltrations in the papillary layer of the derma. Hemotoxylin and eosin, x 88. B. Skin of hand showing the hyalinization and loss of cellularity of the papillary and reticular layers of the derma. Hemotoxylin and eosin, x 48. C. Left triceps muscle. This shows a focal collection of lymphocytes and plasma cells in the damaged muscle. Hemotoxylin and eosin, x 88. D. Right sternocleidomastoideus muscle. Cross-section shows the marked swelling, fragmentation, and hyalinization of muscle fibers and the intense proliferation of the connective tissue in between the muscle fibers. Hemotoxylin and eosin, x 88.*

epidermis were not unusual. The derma, especially in its reticular layer, showed swollen and hyalinized collagenous fibers.

Sections of the gastrocnemius showed little change, although scattered in some fascicles were individual muscle fibers which were swollen, rounded, and deeply stained. The fibrillar striations were unchanged. The sarcolemmal and endomysial nuclei were not increased in number.

Some portions of the left triceps looked essentially normal with an occasional swollen, rounded muscle fiber in the fascicles, but there were areas of intense destruction of muscle fibers. The fibers were swollen or shrunken, hyalinized or fragmented, with loss of striations, accumulations of increased sarcolemmal nuclei, swollen sarcolemmal nuclei, increase in connective tissue, and replacement of lost muscle fibers by connective tissue. A few collections of mononuclear infiltrating cells—lymphocytes and plasma cells—were noted in the devastated muscle (figure 3C). Similar nodules of such cells were seen in the perimysium.

A few swollen, rounded, hyalinized fibers were noted in the right pectoralis major, as well as small collections of infiltrating mononuclear cells in the connective tissue. Otherwise this striated muscle appeared to be intact.

There were changes noted in all portions of the specimen from the right rhomboideus major. In the less involved areas, the changes consisted of swelling of muscle fibers, hyalinization of

portions of muscle fibers, loss of cross-striations, and a marked increase in the number of sarcolemmal nuclei. In more involved areas, these changes were associated with a greater disruption of the muscle fibers and a tremendous increase in the number of nuclei. The most intense change consisted of an intense fibrous overgrowth and replacement of muscle tissue, with a few swollen, hyalinized muscle fibers still contained in the fibrous scar.

The right sternocleidomastoideus displayed the most extensive and consistent pathologic changes. Mild changes consisted of swelling, hyalinization, and fragmentation of muscle fibers with a noticeable increase in sarcolemmal nuclei. There were a few foci of infiltrating cells, mostly lymphocytes. In many areas, muscle fibers were destroyed and replaced by dense, cellular, connective tissue. A few swollen fibers and shrunken or fragmented fibers were found encased in a dense and sometimes hyalinized scar (figure 3D).

Gross examination of the cerebrum, cerebellum, brain stem and spinal cord revealed nothing significant. Portions of the nervous system were fixed in formalin, embedded in celloidin, and stained with hemotoxylin and eosin, cresyl violet, Weil's myelin stain, and Bodian's Protargol.

Sections of the spinal cord, stained for myelin sheath, showed the various tracts to be intact. In the Nissl preparations, the anterior horn cells of the spinal cord were of expected quantity. Nissl bodies were well preserved and there was no evidence of glial proliferation.

In the hemotoxylin and eosin sections of the right common peroneal nerve, there seemed to be no disturbance in the structure of the nerve. The myelin sheath stains showed no demyelinization. With the Bodian stain, the axis cylinders were found to be intact.

Many of the cells of the right inferior cervical sympathetic ganglion contained masses of brown pigment. The nuclei of many of the ganglion cells showed condensation and shrinkage. In the Nissl preparations, the tigroid substance was often destroyed or powdered. One neuron showed two nuclei. The interstitial tissue in the ganglion showed no increase. Such variations have been found in normal autonomic ganglia.

While Adams, Denny-Brown, and Pearson[49] distinguish clinically between acute polymyositis and chronic polymyositis, they recognize that variations in degree, extent, and location of muscle and skin involvement do occur. The diagnosis may best be established by histologic study of the affected muscles which show acute hyaline necrosis of the muscle fiber, marked increase of sarcolemmal nuclei, and inconstant cellular infiltrations. But even such ultimate studies may not be conclusive, and further observations seem to be indicated to delimit these syndromes. At present, no specific therapy is known for these forms.

Progressive Muscular Dystrophy

Nevin,[2] reviewing in 1936 the reports of late progressive muscular dystrophy in the literature, found that 16 of the 27 cases recorded fitted into this category. He pointed out that the clinical and pathologic features of late progressive myopathy corresponded best to Erb's juvenile form of muscular dystrophy. Only two of the certain cases had a family history; all the others were sporadic. Three of the 16 cases had their onset in the fourth decade, 6 in the fifth, 5 in the sixth, and 2 in the seventh decade of life. Nevin reported two of his own cases in detail, one of which had its onset at 59, and the other at 57.

Solomon and Netsky[50] reported two sporadic cases and two families in whom muscular dystrophy began after the age of 30. They pointed out that onset may be as late as 82 years. When the disease begins late in life, clinical features are atypical, but histologic appearance of the muscles in late dystrophy is similar to that encountered in the childhood types.

In reviewing patients admitted to the Neurology Ward of the Hospital of the University of Pennsylvania from 1944 through 1953, 19 patients were found in whom the diagnosis of muscular dystrophy had been suggested. The ages of onset, as far as they could be estimated, were 4 months, 6 months, 1½ years, "childhood," 8, 10, 13, 15, 18, 21, 25, 28, 33, 37, 40, 49, 59, 59, and 60. As can be seen, 6 of these were 10 years or less at onset of illness; 6 were below 30; and 7 were above 30. There was no correlation between sex and age of onset. Three patients were Negroes.

Case 8. E. M., a 64-year-old white woman, was admitted on January 1, 1953. Symptoms had begun at the age of 60 with buckling of the left knee. Then the right knee had given out and pain developed back of the knees, necessitating the use of canes. For the past three years she had been unable to stand.

In 1943, the basal metabolic rate was said to have been plus 46 per cent. An attempted thyroidectomy, at another hospital, had to be stopped because of an alarming rise in blood pressure, and only the right superior pole was ligated. Roentgen therapy of the thyroid gland was given postoperatively.

Examination showed a flaccid paraplegia with movement only in the toes of the left foot, areflexia in the lower extremities, no fasciculations, muscle status unaltered in the upper extremities, sensory system intact for the usual modalities, and cranial nerves unchanged.

Gastric analysis showed achlorhydria with histamine. Queckenstedt test showed no block of the spinal subarachnoid space. Protein of the cerebrospinal fluid was 29 mg. per cent. Blood and cerebrospinal fluid syphilitic tests were nonreactive. A tracer study with I^{131} revealed 23 per cent take-up in forty-eight hours. Protein-bound iodine was 5.8 micrograms per 100 cc. of blood serum.

Electrical stimulation of the muscles of the lower extremities gave responses only in the muscles of the right foot, which could still be moved voluntarily. The electromyogram showed widely separated responses.

Biopsy of the right quadriceps femoris showed decided involvement of muscle fibers. There were many swollen, rounded muscle fibers and many small fibers in a mass of connective tissue and fibers were frequently hyalinized. Sarcolemmal nuclei were abundant, of varying sizes and shapes, often surrounding a muscle fiber like a rosette. Some fatty infiltration was noted in the muscle fascicles.

Case 9. D. M., a 62-year-old white male, was admitted on August 23, 1951, with a history of onset at age 59. He had first had pain in the joints of his left lower extremity. Weakness and heaviness of the right lower extremity began eight months before admission. About the same time, he noted pain and weakness of his right upper extremity. Stair climbing was difficult and he used his arms to lift himself out of a chair. He was still working as a cabinetmaker. In 1935, he was said to have had a thyroid condition, for which he received treatment for several years.

Examination showed atrophy of right rhomboids; right winged scapula; atrophy of right deltoid, biceps brachii, and possibly of left deltoid; weakness of abduction of right arm and flexion and extension of right forearm; smaller left gastrocnemius; and weakness of hip flexion and abduction, particularly on the left. He had difficulty stepping up on a chair and rising from a supine position. He could walk well on toes and heels. There were no fasciculations, no alterations in reflexes, and no pathologic reflexes. Vibration-sense was impaired in the feet, especially on the right. Cranial nerves were intact.

Queckenstedt test showed no block of the spinal subarachnoid space. Protein of the cerebrospinal fluid was 25 mg. per cent. Blood and cerebrospinal fluid syphilitic tests were nonreactive. Basal metabolic rate was minus 11 per cent. Protein-bound iodine was 4.8 micrograms per 100 cc. of blood serum.

Electrical studies of the atrophic right infraspinatus muscle showed a chronaxie of 0.4 millisecond. The electromyogram of this muscle showed no fasciculations and normal motor bursts on voluntary effort.

Various treatments were tried including a course of wheat germ oil. When seen on November 20, 1953, he was still complaining of pains in his extremities, but had less weakness in the lower extremities. Muscle wasting was more evident in the upper extremities.

Case 10. K. R., a 63-year-old white woman, was admitted on January 15, 1948. She had first noted increasing difficulty in climbing stairs at the age of 59 and since then had frequent falls. After falling, she was unable to get up without help, yet had no difficulty in walking.

No muscle wasting was evident on examination. The patient was unable to rise from a sitting position on the floor, and could not get out of a low chair without using her upper extremities. Right thigh and leg muscles showed more weakness than those of the left. Gait was steady. Patellar reflex was absent on the right and impaired on the left. There were no pathologic reflexes. Sensory system was intact and cranial nerves were unchanged.

Queckenstedt test revealed no block of the spinal subarachnoid space. Cerebrospinal fluid pressure was 220 mm. of water and protein was 45 mg. per cent. Syphilitic tests were negative. Blood serum creatinine was 0.7 mg. per cent. Biopsy of left gluteus maximus muscle showed some muscle fibers to be larger than normal, but it was felt that this specimen did not show any diagnostic changes.

Tests in February, 1953 showed creatinine excretion in urine to be 0.43 gm. per twenty-four hours; 0.55 gm. per twenty-four hours; and 0.53 gm. per twenty-four hours. Creatine excretion was 0.43 gm. per twenty-four hours; 0.25 gm. per twenty-four hours; and 0.73 gm. per twenty-four hours. Body weight was 156 lb.

When seen on January 26, 1954, the patient

was unable to get upstairs or to get out of her chair. Both knee jerks were absent.

Case 11. J. H., a 50-year-old colored woman, was admitted on April 6, 1948. The illness had begun when she was 49, with pains in the upper extremities, swelling and stiffness of fingers, and stiffness of knee joints. These conditions improved with treatment, but she began to lose weight and noticed generalized weakness.

Although wasting of muscles was uncertain, they exhibited a generalized weakness, more so of the proximal musculature of all four extremities. The patient was unable to raise her arms above shoulder level and, when placed on the floor, she could not sit or stand. She could bend forward and backward at the waist, but was unable to step onto a chair. No fasciculations were present and no change in reflex activity. Sensory system was intact. Cranial nerves were unchanged.

Queckenstedt test showed no block of the spinal subarachnoid space. Cerebrospinal pressure was 150 mm. of water; protein was 20 mg. per cent. Tests for syphilis were negative.

Faradism was unchanged in the muscles of the face and upper and lower extremities. Biopsy of the left quadriceps femoris showed pale and greatly swollen muscle fibers, hyalinized muscle fibers with loss of striations, and many groups of sarcolemmal nuclei in clusters resembling giant cells.

These four cases of late developing myopathy are included in this group with the realization that they may fit into some of the other groups after more extensive study. Case 8 was for a long time considered to be one of late muscular dystrophy, but the skin changes and the cellular infiltrations of the muscle led to a correct diagnosis of some form of dermatomyositis or polymyositis. The changes noted in the biopsy specimens in cases 8 and 11 were suggestive of those encountered in polymyositis. Case 10 fits clinically into the group known as menopausal muscular dystrophy. In none of the cases were the clinical features typical of muscular dystrophy. There was no family history of such disease. These reports emphasize the diagnostic problems encountered in such cases and the present uncertainty of this whole group.

The clinical picture of muscular dystrophy is one of slowly progressive weakness of striated musculature. There is no associated sensory disturbance. The weakness is followed by a gradual muscular atrophy. There are no fasciculations. There is a tendency for the muscles to be involved fairly equally on both sides and the locale of early involvement seems to be the shoulder girdle and arm muscles. Apparently in the late form, false muscular hypertrophy does not occur. The wasting and weakness may spread gradually to involve the muscles of the trunk, lower extremities, and face.

In these patients, the deep and periosteal reflexes of affected muscles become impaired and finally absent. The chronaxie values remain unchanged as long as any response is available from a diseased muscle. The electromyogram reveals no fibrillations and no fasciculation potentials.

The chemistry of the blood and especially of the urine has some diagnostic value in muscular dystrophy.* Blood serum creatine may be increased; blood serum creatinine is unchanged. The urine creatine is increased; urine creatinine excretion is diminished. The creatinuria is found in other conditions and is normal in childhood, pregnancy, hyperthyroidism, chronic arthritis, poliomyelitis, Addison's disease, and others. Hence, the decreased urinary excretion of creatinine has been felt to be more significant.[51] Glycine given by mouth temporarily increases the creatinuria.[52] A creatine tolerance test has been described as significant in muscular dystrophy,[53] but has been denied by others.[51] The test consists of administering 1 to 3 gm. creatine by mouth. In normal people, this creatine is retained, while in patients suffering from muscular dystrophy most of the creatine is excreted in the urine.

Biopsy of affected muscle often has diagnostic value. According to Hassin,[51] the histologic changes in dystrophic muscle "are in the form of atrophies, hypertrophies, loss of striations, myofibrillary disruption, obliteration of Cohn-

heim's fields, hyalinization, fatty metamorphosis (lipomatosis), all resulting in a formation of a connective tissue scar (fibrosis)." Sometimes, it is necessary to study the nerve fibers and motor endplates in such specimens to eliminate muscle atrophy of neural origin.[55]

The treatment of muscular dystrophy is still most unsatisfactory. No specific therapy exists, for the etiology is obscure. Wheat germ oil or the synthetic tocopherols seem to have little value despite suggestions to the contrary.[5]

Miscellaneous Muscular Disorders

The wasting of muscle and the impairment of power during the senium is well known. The senile wasting of muscles is gradual and generalized. Loss of muscle power is not as severe as would be expected from the loss of muscle volume. Senile muscular atrophy is apparently part of the general senile loss of parenchymal tissue. Pathologically,[56] there is a reduction in the size of muscle fibers, a pigmentation at the poles of the muscle nuclei, fatty degenerative changes, and so forth. The similarity in the histologic appearance of senile muscle to that seen in chronic malnutrition has been noted.[56]

Bramwell[57] described two patients whose muscle weakness began at the ages of 53 and 59 years respectively. A similar case was reported briefly by Denny-Brown,[58] but symptoms in his patient began at the age of 37. The essential features of this syndrome consisted of a progressive wasting and weakness of the quadriceps femoris muscles and loss or impairment of the knee jerks. These cases seemed to present a picture compatible with a primary muscle disease. Denny-Brown[58] stated his patient improved by taking 5 gm. glycine twice daily.

Shy and McEachern[6] described a group of 12 patients, consisting of 11 women and one man, who exhibited a progressive weakness of the hip and shoulder girdle muscles, which they called menopausal muscular dystrophy. Process was slow or rapid. Their patients had trouble rising from a chair or ascending stairs, as well as raising their arms above their heads. Muscles in hip and shoulder areas were found to be weak, but not often wasted. Reflex changes were slight. The importance of this group of cases is that they were supposed to respond satisfactorily to wheat germ oil or administration of cortisone.

The weakness and wasting of muscles late in life are still the source of considerable discussion and uncertainty. Adams, Denny-Brown, and Pearson,[59] for instance, regard the late forms of muscular dystrophy as reported by Nevin,[2] the quadriceps weakness as reported by Bramwell,[57] and the menopausal muscular dystrophy of Shy and McEachern[6] as a form of "chronic polymyositis occurring during the climacterium or late years of life."

Of considerable significance in later maturity is a dawning association of carcinoma, especially of the lung, with various neurologic disorders.[60-63] Sensory neuropathy, polyneuritis, subacute cerebellar degeneration sometimes with dementia, and myopathy have been found associated with carcinoma. Some of the myopathies have presented myasthenic[62, 63] and even myotonic[63] features. Neurologic symptoms have often preceded cancer symptoms or even diagnosis of the carcinoma by many months. The central nervous system symptoms were not due to secondary metastases. The muscular wasting and weakness was beyond that encountered in carcinoma-

*Normal range of blood serum creatine is estimated as 0.2 to 0.9 mg. per cent and that of blood serum creatinine as 0.4 to 1.4 mg. per cent. Normal excretion of urine creatine is estimated at about 2 to 3 mg. per kg. of body weight; for example, a 150-lb. man would excrete in his urine .136 to .204 gm. creatine per twenty-four hours. Normal excretion of urine creatinine is estimated at about 22 to 23 mg. per kg. of body weight; for example, a 150-lb. man would excrete in his urine about 1.5 gm. creatinine per twenty-four hours.

tous cachexia or malnutrition. Henson and associates[62] felt that the atrophic paresis of carcinomatous myopathy was most marked in the limb girdles and proximal parts of the limbs and that such a clinical picture was sufficiently constant to make their recognition a simple matter. While the myopathic or myasthenic disturbances could occur alone, it was not unusual to find signs of peripheral neuropathy or involvement of the central nervous system combined in certain cases. Remissions have been observed in the neuromuscular disorders associated with carcinoma.

The etiology of the neuromuscular disorders associated with carcinoma is obscure at present. There seems to be no direct causal relationship. Barraquer-Bordas and Lowenthal[61] suggested that the neural and muscular abnormalities are due to a carcinotoxin. A virus was considered a possible cause in 2 of Henson's 19 cases.[62] The possibility that these may represent a disorder of vitamin E or pantothenic acid[60] metabolism seems likely especially since disorder of the pyruvate tolerance test has been noted in such patients.[62] Treatment is not specific, but is essentially directed toward the carcinoma. Henson[62] stressed the use of neostigmine to relieve the myasthenic symptoms. Adequate doses of vitamins, especially B and E, are suggested at our present state of knowledge.

Heathfield and Williams[63] stated that in all patients in the later age groups who show signs of a peripheral neuropathy or myopathy, carcinoma of the lung should be searched for by x-ray and bronchoscope. Brain, Daniel, and Greenfield[64] and Lennox and Prichard[65] stated that improvement followed removal of the growth, although Henson and coworkers[62] could not find such a correlation in their studies.

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The nutritional status of noninstitutionalized aged persons

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Of a group of 53 noninstitutionalized persons over 60 studied for nutritional deficiencies, almost 95 per cent had signs of vitamin deficiency; one-half of 49 had lower ascorbic acid content than the accepted minimum; 5, a lower vitamin A content; and 4, a lower carotene content. Forty per cent had a deficient calorie intake and an equal proportion, deficient proteins. The diet of one-half to two-thirds was deficient in essential vitamins, iron, and calcium.

◆ One of the vital factors in maintaining good health in older persons is adequate nutrition. Much has been written about the nutritional needs of the aging person but most of the studies that have been conducted have been of older persons dwelling in institutions.

An institution presents the easiest opportunity for studying nutritional needs but institutionalized persons are a selected group. The nutritional status of the inmate is influenced, among other factors, by the diet and physical care provided and the number of years of residence in the institution. In addition, institutionalized persons have a higher incidence of debilitating diseases than have individuals living at home. They also comprise a small proportion of aged persons since only about three per cent of such persons are institutionalized.[1] Few nutritional studies have been conducted of older persons living at home, and there is little actual evidence of the nutritional status and food habits of this large number of aged persons.

It is of interest to review the findings by several investigators of the nutritional status of institutionalized aged persons. Jolliffe examined 20 persons in a hospital and home for the aged in New York City and found that only 2 persons, or 10 per cent, showed clinical signs suggestive of active nutritional deficiency.[2] In the same institution Rafsky and Newman analyzed the diets of 65 persons over 65. The food intake was weighed and the total calories, protein, fat, and carbohydrate content calculated. An analysis was also made of a twenty-four-hour intake of thiamine, riboflavin, carotene, vitamin A, niacin, ascorbic acid, and iron. The diet was found adequate in carbohydrate, fat, protein and total caloric intake and no relationship between diet and laboratory evidence of nutritional disease was discovered. In previous studies, they reported a need for higher than accepted amounts of vitamins B and C to achieve a constant saturation point, maintaining that larger amounts were necessary because of poor absorption and assimilation.[4, 5]

Pyke and co-workers evaluated the nutrient content of diets eaten by 10 old women living at home, 18 in almshouses doing their own housekeeping, and 3 groups living in institutions in London.[6] The intake of vitamins A and C was

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found to be low but calcium, iron, and vitamin B intake was considered reasonably adequate based upon accepted British standards. It should be noted that the British standard requirements are lower than the recommended daily allowances for specific nutrients of the Food and Nutrition Board of the National Research Council in the United States.

Ohlson and her associates analyzed the recall diets of 100 noninstitutionalized women from 40 to 78 years of age and found that they averaged from 300 to 500 calories less than representative samples of young women, and that 30 women in the group had a mean caloric intake of less than 1500 calories per day. This lower average caloric intake of the older women is in accordance with the physiologic decrease of energy output of older people. The low caloric intake tended to be associated with a low consumption of protein and, it is also assumed, low consumption of thiamine, iron, phosphorus, and possibly riboflavin, when judged according to the higher caloric intake.[7]

Methods and Findings

The present study was undertaken in order to determine the nutritional status of older persons living at home. The study had a two-fold objective: to evaluate the food habits and nutritive status of a group of persons over 60 and to plan an educational program in nutrition based upon clinical and other findings. The study was conducted by members of the staff of the Nutrition Clinic of the Lower East Side Health Center of the Department of Health of New York City. The subjects of the study were members of the Sirovich Day Center for the Aged, an institution operated by the Sirovich Relief Society and the Department of Welfare of New York City. The center provides social, cultural, recreational, and educational facilities for approximately 600 persons over 60.

The study included a group of 53 persons chosen at random and consisting of 27 men and 26 women. Half of the group was between 60 and 69 and the other half was over 70 as shown in table 1.

TABLE 1
AGE DISTRIBUTION OF 53 PERSONS OVER 60

Age group	*Male*	*Female*
60-64	2	10
65-69	9	5
70-74	8	4
75-79	5	4
80-84	1	2
85-94	2	1
Total	27	26

Of this group, 36, or 68 per cent, were receiving public assistance, 5 were supported by their children, 5 were living on their savings, and 2 had savings and received supplementary assistance from their children. The remaining 5 had no regular means of support. Of the entire group, 39, or close to 74 per cent, lived alone. Twenty-seven had nearly all their meals in restaurants and 25 never ate out.

There were three phases to the study:

1. A physician of the Nutrition Clinic of the Lower East Side Health Center obtained a medical history and performed a physical examination of each member of the group.
2. The Bureau of Laboratories of the Department analyzed the blood of 49 persons for vitamin A, carotene, ascorbic acid, iron, and protein.
3. A nutritionist of the Nutrition Clinic interviewed each subject to obtain a twenty-four-hour recall of food intake and a social and economic history.

Clinical signs[8] suggestive of nutritional abnormalities were observed in 50 of the 53 individuals. Three showed no clinical signs of nutritional deficiencies, and some had more than one of the clinical manifestations (table 2). Twelve of the persons were considered obese, while 7 were underweight.

The blood of only 49 of the 53 members of the group was examined for vitamin A, carotene, ascorbic acid, iron,

TABLE 2

CLINICAL SIGNS OF NUTRITIONAL ABNORMALITIES IN 50 OLD PEOPLE

Clinical signs	*Number of persons*	*Clinical signs*	*Number of persons*
Glossitis	36	Folliculosis	8
Gingivitis	21	Blepharitis	8
Corneal arcus	17	Xerosis	7

and protein. More than 50 per cent of those whose blood was examined had a lower ascorbic acid content than the accepted normal minimum.[9] Five had deficient vitamin A; 4, deficient carotene; and 1, a low iron content. None manifested a low protein content in the blood (table 3).

Evaluation of the caloric intake as illustrated in table 4 reveals that 6 individuals had a diet more than 20 per cent above the modified recommended allowance and 26 persons, or approximately 50 per cent, had a diet within 20 per cent of the modified recommended allowance. Thus 21 or 40 per cent had an intake less than 20 per cent of the allowance and may therefore be considered as having had a deficient caloric intake. The recommended allowances were based upon the average calorie requirements, taking into consideration the energy requirement, age, height, and sex as suggested by Jolliffe.[10]

The twenty-four-hour diet was also evaluated as to protein, calcium, iron and vitamin content, according to the methods and standards of the Nutrition Clinics of the New York City Department of Health. They were compared with the recommended allowances for specific nutrients of the Food and Nutrition Board of the National Research Council, 1948. A satisfactory diet was considered one that contained 80 per cent or more of the recommended allowance (table 5).

Of the 53 diet records studied, about 40 per cent were deficient in protein; 60 per cent in calcium; 75 per cent in iron and thiamine; and about 50 per cent in vitamin A, riboflavin, niacin, and ascorbic acid. Although the proportion of deficient dietary constituents was approximately the same in men and women, the proportion of men who had a deficient ascorbic acid intake was twice that of women. Only one-half of the women had an inadequate ascorbic acid intake, while two-thirds of the men were in that category.

TABLE 3

BLOOD FINDINGS OF 49 PERSONS OVER 60

Finding	*Number of persons*
Ascorbic acid	
over 0.7 mg. %	19
under 0.7 mg. %	29
under 0.6 mg. %	22
under 0.4 mg. %	15
0.1 mg. % or less	1
not determined	1
Vitamin A	
over 30 mcg. %	42
under 30 mcg. %	5
under 20 mcg. %	2
not determined	2
Carotene	
over 50 mcg. %	43
under 50 mcg. %	4
not determined	2
Serum iron	
over 45 mcg. %	45
under 45 mcg. %	1
not determined	3
Total protein above 6.0%	49

TABLE 4

24-HOUR CALORIC INTAKE OF 53 PERSONS OVER 60

Calorie intake	*Male*	*Female*
Over 20% modified recommended allowance	3	3
Within 20% modified recommended allowance	15	11
Below 20% modified recommended allowance	9	12
Total	27	26

Comments

The nutritional status in older persons cannot be accurately evaluated by one method of examination. There is fre-

TABLE 5

EVALUATION OF ONE-DAY DIET RECORDS OF 53 PERSONS OVER 60

Number of subjects	*Protein*	*Calcium*	*Iron*	*Vitamin A*	*Thiamine*	*Riboflavin*	*Niacin*	*Ascorbic acid*
Satisfactory diet								
Male	18	12	8	12	8	12	11	9
Female	14	8	5	12	6	12	13	18
Total	32	20	13	24	14	24	24	27
Unsatisfactory diet								
Male	9	15	19	15	19	15	16	18
Female	12	18	21	14	20	14	13	8
Total	21	33	40	29	39	29	29	26

quently a discrepancy between the diet consumed and the clinical and blood laboratory findings. Nutritional deficiency is not always due to a deficient diet but may be the result of conditioning factors, such as interference with proper absorption of foods. There have been consistent reports of a high incidence of low blood ascorbic acid values in older persons, which may represent poor absorption from the intestinal tract. The low ascorbic acid values have also not been associated regularly with clinical signs of scurvy. In this study, of the 23 subjects with blood ascorbic acid levels below 0.6 mg. per cent, 12 had gingivitis. Of the 19 with gingivitis who had blood level determinations, 11 had blood levels below 0.6 mg. per cent.

Three types of analysis were conducted in this study: physical examinations, blood tests, and diet evaluations. While the findings from one type are not necessarily indicative of dietary deficiency, the summation of findings in the three types is more conclusive. Over half the group studied showed an inadequate vitamin intake. Clinical findings suggested vitamin deficiency in almost the entire group, and laboratory studies indicated low blood, ascorbic acid (0.6 mg. per cent) in about half of the group. The total findings may be considered as evidence that the noninstitutionalized ambulatory aged, as judged by the sample studied, does not consume an adequate diet, particularly in vitamin C.

These findings, together with the socioeconomic factors ascertained, indicate the need for a practical nutrition education program for this group. Such a program is now being conducted at the Sirovich Day Center and among other aged groups in the Lower East Side district and will be reported elsewhere.

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Management and prognosis of suicidal attempts in old age

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◆ At least 80 per cent of suicidal old people over the age of 60 are suffering from depressions.[1] All suicidal old people, no matter what the reaction type may be, are depressed. If the individual is suffering from an organic dementia, his deterioration is usually not far advanced and affect and volition are retained. A typical case is described briefly.

A. B., a 90-year-old widower, attempted suicide by cutting his throat. There was no family history or previous personal history of nervous or mental illness. He lost his mother when he was a small boy, was unhappy with a stepmother, and ran away from home when he was 12 years old. However, he settled down, married happily, had a large family, and was a steady worker throughout his life. He was a man of limited interests and few friends. In his ninetieth year, with hearing and sight impaired, he was confined to the house. He became depressed, preoccupied with his bowels and his bladder, complained of headache and of vertigo, slept poorly, and lost his appetite for his food and for his pipe. Since the daughter with whom he was living was at work during the day, he was left much alone and he felt lonely. Shortly after his ninetieth birthday, he began to say he wished God would take him away. His doctor confined him to bed and he reacted very badly to this, feeling useless and a bother to his relatives. A fortnight after he was put to bed he attempted suicide. In the hospital to which he was taken, he said that he regretted that he had not succeeded in killing himself.

This case is quoted to emphasize that in Britain suicides occur at a higher rate in men over 65 than in any other age and sex group, and may occur even in extreme old age. The case was typical in its features. The old man was suffering from a severe depressive state, he had come from a "broken home," and his previous personality had been rather rigid and asocial. In old age he had become physically enfeebled. Then feelings of loneliness, uselessness, and of being a burden upon others, and fears and preoccupations with bodily dysfunctions developed—these are the emotions which commonly precede these suicidal acts.

At least 80 per cent of those who attempt suicide in old age are suffering from depressions. A follow-up study has shown that nearly two-thirds of these depressives may be expected to recover, but that 20 per cent of the recovered may relapse and that a further 15 per cent may die of physical illness within two years. Twelve per cent of those who attempt suicide may be expected to complete the act within two years. After a suicidal attempt, suicidal risk in old age is therefore considerably greater than it is among those who attempt suicide at earlier ages.[2] Any relapse of the old person into depression must be treated urgently and with full recognition of this serious suicidal risk.

Management

Although suicidal gestures are not uncommon among younger people, particularly those of chronically unstable temperament, they are rare in old people.

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Any suicidal act by an old person must therefore be treated with grave concern. The patient should be admitted initially to the observation ward of a general hospital. The results of a suicidal attempt usually require skilled surgical and medical treatment and time must be found for a comprehensive and unflustered review of the etiologic factors and the social setting. In this review, the physician, psychiatrist, and psychiatric social worker should collaborate.

Apart from the injuries they have inflicted upon themselves, about 60 per cent of these old people suffer from significant physical disease, most often of the cardiovascular system. Adequate treatment of heart failure, of malnutrition, or of drug intoxication, may effect a considerable and even rapid improvement of the mental state, particularly where there has been some clouding of consciousness.

The psychiatrist is particularly interested in whether, in addition to the obvious affective disorder, there is evidence of organic dementia, since the prognosis for recovery depends largely upon this. It may be some time before he can be sure. An old person suffering from a depressive psychosis may be confused and give the impression even of being demented. This confusion may clear in a few days after the suicidal attempt, unmasking intact mental faculties. It is normally possible to classify the reaction type in these suicidal cases, since the individual is usually mentally involved to the extent of psychosis.

The social worker must investigate the home situation in detail—living conditions, financial status, availability, capacity, and willingness to help of relatives and friends. The social worker also plays an important role in promoting understanding and sympathetic attitudes among those to whose company the patient may return and in continuing a friendly contact with these often lonely people after their return home. If the social environment seems to have fostered the old person's suicidal attempt, every effort must be made to improve that environment before he returns to it.

Though one is naturally reluctant to recommend admission of an old person to a mental hospital, about one-half of these cases require further care and treatment in such an institution. There are two main causes of this: either the depressive reaction is of such severity that the patient cannot be adequately treated or safeguarded at home, even when the home is satisfactory; or the home situation is so unsatisfactory that he cannot be sustained where he had been living. With regard to treatment, it is sufficient to say here that old age in itself does not exclude any treatment which helps younger depressives.

Prognosis

Follow-up study was made of 40 consecutive cases of people aged 60 years or over who attempted suicide and were admitted to a general hospital.[1] The situation found two years after the attempts had been made revealed that 7 of the 40 had been suffering from states of organic dementia or from long-standing psychopathic or paraphrenic reactions. These intractable conditions were found to have persisted. On follow-up, one individual was found to have had the wrong diagnosis made originally, when she was said to be suffering from an organic dementia.

Of the 40, 33 had been suffering from depressive states. Twenty of these, or 61 per cent, recovered—the expected recovery rate for depressives in this age group. Of those who recovered, 20 per cent had subsequent depressions and a further 15 per cent died of physical illness.

Of the whole group of 40, 13, or 32.5 per cent, died within the two years following their suicidal attempt. Five of these, 3 men and 2 women, or 12.5 per cent of the whole group, committed suicide. These 5 individuals were still in

their sixties: 3 were manic-depressives who had recurrences; one was an involutional melancholic who relapsed after apparent recovery; and one was a psychopath with depressive swings of mood. In 4 of the 5 cases, the same method of suicide was used as had been chosen previously, while 1 man, who had previously taken aspirins, drowned himself and thus identified himself with his father who had committed suicide in that way. In 3 of the 5 cases, the setting was identical: one of the women killed herself on the second anniversary of her suicidal attempt. The motives for these suicides seemed to be the same as those for the suicidal attempts which had preceded them—loneliness, fears of insanity and of mental hospital admission, intolerance of physical incapacity, and, in one case, guilt over homicidal impulses. In this age group, the rigidity of the suicidal pattern is notable and should facilitate the prevention of further suicidal attempts. Two, if not 3, of these 5 suicides were probably preventable.

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CONJUNCTIVITIS in the infant and the aged presents a syndrome quite different from the disease in young adults. In a twenty-year epidemic of membranous conjunctivitis in Japan, the disease—an infantile form of epidemic keratoconjunctivitis—had characteristic systemic complications: high fever, angina, pharyngitis or otitis media, and diarrhea and vomiting. Bacterial or inclusion conjunctivitis was seen only in young children.

Membrane formation and degree of systemic reactivity are high in infancy, decrease to a low at age 25, and thereafter increase with age. On the other hand, follicle-forming and preauricular node and corneal reactivity are not found under 2 months of age, increase to about age 25, and thereafter decrease with age. Diet rich in fat and protein appears to increase the fibrin reactivity.

Diphtheria toxoid is used to treat membraneous conjunctivity. Its effect is direct and not connected with an immunization process.

The difference in clinical findings in conjunctivitis in the various age groups is related to the changes in conjunctival epithelium occurring with age. In trachoma and in conjunctivitis caused by Hemophilus conjuctivitidis the different clinical picture between infants and adults was also observed.

Y. MITSUI, C. TANAKA, and K. YAMASHITA: Change in the constitution with age. Its influence on the clinical symptoms of conjunctivitis. Am. J. Opth. 39: 540-546, 1955.

Professional opportunities for the older nurse

KATHARINE J. DENSFORD, R.N., D.Sc.
MINNEAPOLIS, MINNESOTA

We must change our concept of the older nurse, as of any older professional person, considering her a resource for the nation, not a problem. She, in her turn, must develop a habit of study and retain a flexible attitude toward new knowledge. Depending upon her interests and abilities she may embrace opportunities for service in any field.

◆ Before me lie two documents. One of these, Facts About Nursing, 1950, includes a center chart entitled Opportunities in Nursing. Dozens of different kinds of positions are listed, including the main classifications of institutional service by staff nurses, head nurses, supervisors, and directors in civilian and governmental hospitals and related agencies; public health nursing in private and publicly supported agencies; nursing in private practice; nursing education in schools and service agencies, both institutional and public health; executive and administrative positions; and other positions, such as those in industry, doctors' offices, and transportation.

No statement occurs on the above chart that could possibly be construed to mean that these opportunities are closed to the older nurse. In fact, there is no reference to age.

The other document, one of many in the study of aging, is the Report of a Project on the Older Nurse, conducted in 1954 by the Professional Counseling and Placement Service of the American Nurses' Association. The older worker is defined in this report as an adult who meets with resistance to employment, continued employment, or reemployment, solely because of age.[1] Interesting is the comment that the older nurse cannot be singled out as being any specific age. She may be anywhere from 45 to 65 or more years old.[2]

My main thesis – that the so-called older nurse can really embrace all opportunities in nursing right across the board, be the field what it may, and that the older nurse constitutes one of the greatest resources of the profession and, healthwise, of the nation–gains strong support from these two documents. In the listing of the myriad opportunities in nursing no age limitation is indicated and in the recent study of the older nurse it seems impossible to differentiate her by the criterion of age alone. That the older nurse forms a large reservoir of nurse power may be cited in one large hospital where 90 per cent of the nurses who apply to it for employment are 40 or more.

Special Opportunities for Service

As a volunteer the older nurse may make a real contribution–for example, in teaching home nursing, in participating in library activities, in the keeping of records in various types of agencies, and in assisting in fields related to research. For these functions she may also be paid.

As a paid worker she should be able to participate in any and all areas of nursing, depending upon her interest and

KATHARINE J. DENSFORD *is professor and director, University of Minnesota School of Nursing.*

ability. She can often provide many much needed services: week-end nursing, particularly in institutions; evening and night assignments in institutions, including hospitals and nurses' infirmaries; office nursing; hostess services in institutions, nurses' residences, doctors' offices; health services in public schools, colleges, and universities; nursing in industrial plants; private patient nursing; care of supply departments; and consultation service and professional writing.

In part-time employment, the older nurse, visualized as an educational member of the staff, has potential for tremendous service. She might be what Mrs. Bertha G. Byrne recently referred to as a "ward visitor." In many of our hospitals today, patients often feel they see only the nursing aides, because duties removed from the bedside occupy the time of doctors and nurses. These patients would welcome an older nurse with time to visit them and to explain to them and their families the implications of treatments, operations, and medications. The older nurse can often offer psychologic rehabilitation in ways which a less experienced person may not do.

Attitudes and Adaptability

What are some of the factors contributing to the continuing service of the older nurse to society? Achievement, success, and satisfaction in any nursing effort seem to depend in large part upon factors not related primarily to age. Among those stressed in the Report of a Project on the Older Nurse as probably most important is the development of a habit of continuous study and a flexible attitude toward new knowledge. "The well-qualified nurse," the report states, "is always in demand regardless of age."[3] The older nurse should be near her job so that she does not need to spend too much time nor effort in travel. She should be removed as a competitor of the younger nurse and appointed usually to hours when the competition may not be so great and also to part-time positions, of which there are hundreds of types.

Inherent in such continuing service on the part of the older nurse is a basic concept of the aging expressed in a personal memorandum by Dr. Halbert L. Dunn, Chief of the National Office of Vital Statistics of the Public Health Service. Dr. Dunn says, "The principal problem of the aging is to change the concept that aging *is* a problem for society to the concept that old age is a period of great value to society. The concept that the older, postmature person has a major role to play in society comes from the realization that the older group of persons represents the greatest single human resource available to the nation which could be dedicated, as a national contribution, toward the development of a creative future for mankind as a whole. In this age group, there exists the experience, resourcefulness, wisdom, and leisure to transform our world if the potential force it represents is unleashed through a dynamic and dramatic concept. Such a concept is a challenge which would transform old age into a period of adventure and 'worth-whileness.' As a by-product of such a revitalized older population, a considerable percentage of the problems of old age—neurosis, debility, chronic disease, invalidism, lack of economic independence, and the maladjustments of living—would be eliminated."

The development of this concept of older age as a time for creative endeavor and social usefulness would bring into focus the emphasis upon every person, including the older nurse, doing something worthwhile and making of older age not a problem, but an opportunity.

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Editorial

Geriatrics—potential unlimited

THE FACTS and statistics pertaining to the increasing longevity of our population are well known. We are all aware that greater age also increases the possibility of multiple pathologic processes in the individual, with associated difficulty in proper management and treatment. The resulting medical, economic, psychologic, and social problems presented by the elderly are, therefore, demanding more and more attention throughout the country.

Aging may be considered as that larger component which frequently includes concomitant chronic illness and severe disability. They may exist in the same patient simultaneously and in any number, combination, or degree of severity. Aging would appear to be that catalyst which brings about a reaction of hopelessness; the net result is frequently one of absent or, at most, passive action in the direction of rehabilitation and readjustment of the patient. On the other hand, the existence of senescence alone is often the major cause of disablement of the individual, both in regard to himself and to society. The further insults of forced retirement and the feeling of uselessness cause increased deconditioning and demoralization.

Even though we are dealing with problems of aging, medical science is constantly developing new approaches and technics, which give us an outlook of understanding and hope in solving the complex difficulties of our older citizens. In addition, the steady increase in longevity has led to a new look at the process of aging. We are studying the relationship of functional and physiologic age to chronologic age. We believe that, along with the improvement of functional capacity for the older individual, purposeful activity and exercise to the point of physiologic and mental limitations offer considerable potential for health maintenance and useful living. Physical medicine and rehabilitation, as well as other medical specialties, are making a definite contribution in the coordinated team approach for restoration of the older patient. Physical medicine and rehabilitation principles applied to prevention, treatment, health maintenance, and rehabilitation of geriatric illness and disability have given promising results. Adequate rehabilitation counseling, activities of daily living, reconditioning procedures within physical tolerance, and medical guidance for selective placement in employment, when indicated, are all important facets of this work.

The conditions which afflict aging patients offer especially favorable clinical opportunities for use of physical medicine and rehabilitation methods. Through a dynamic program of rehabilitation, we can help these patients to achieve a fuller life of comfort, happiness, and usefulness. Coincidentally, it has been shown that a rehabilitation program for geriatric patients pays off economically in making many of these individuals self-supporting

and for many others there is the possibility of partial or full employment. In either event, a custodial patient becomes independent and is given the opportunity of contributing to society.

With further progress and full implementation of the team approach to geriatric problems, the human life span can be considered as a continuous experience in which youth, adulthood, middle age, and old age merge smoothly into one another, and become integrated parts rather than separate entities. In this framework, old age assumes the proportions of a dynamic and developmental life experience, and the discipline of geriatrics becomes in a sense a science of hope and promise.

A. B. C. KNUDSON, M.D.
Washington, D. C.

The duration of widowhood

THE present great lengthening of life has resulted in a considerable extension of the average period during which husband and wife can live together. Half a century ago, some 28 out of 1,000 marriages were broken each year by the premature death of husband or wife. Now, this figure is nearer 18 in 1,000. With the length of life now increasing more rapidly for women than for men, the number of widows is increasing more rapidly than is the number of widowers. According to the Statistical Bulletin of the Metropolitan Life Insurance Company for September 1953, some 660,000 marriages are broken by death each year, and in two-thirds of the cases the wife is the survivor.

As one would expect, the probability that a wife will be left a widow increases with the number of years by which she is younger than her husband. For instance, when the husband and wife are of the same age, the chances that the wife will outlive her husband are 60 per cent, but the figure rises to 70 per cent when the husband is five years older, and it rises to almost 80 per cent when he is ten years older.

Some 3 in 4 of the women who are widowed by 50, and some 9 in 10 of those who are widowed by 40, will go on living for twenty years. Even at 65, half of the widows can expect to survive for fifteen years, and 1 in 3 is likely to live twenty more years.

This tendency to early widowhood and long survival poses a serious problem to thousands of women, and especially to those left with children. The women widowed late in life are not so likely to have trouble with dependent children, but those without sufficient income face the serious problem of getting employment when they are older. For an elderly widow the chances of marrying again are slim.

All of this means that a wise and kindly husband, if he has a fair income but no large amount of savings, should always carry insurance adequate to take care of his wife when he dies.

WALTER E. ALVAREZ, M.D.

Digests
FROM CURRENT LITERATURE

Age Distribution of Cancer

H. GROSSE. Ztschr. f. Altersf. 8: 244-254, 1955.

Establishing the age incidence of cancer in general or cancer of certain organ systems may prove misleading statistically. The fact that a certain type of cancer is seen most frequently in a certain age group does not necessarily mean that its incidence decreases beyond that age. The relative incidence of cancer may be greater in an older and consequently smaller population than it is in the age group of apparent maximum incidence.

A further source of error may be introduced by the selection of information on which the statistics are based. The accurate diagnosis of cancer, made at postmortem examination, is frequently based on material from large teaching hospitals. Such a hospital population is heavily loaded with severe illnesses like cancer. Statistics based on death certificates are weighted with all the diagnostic errors of such documents. In addition, there is no practical way to account for those cancers that may have been cured by timely intervention.

If cancer mortality, in terms of deaths per 10,000 living, is plotted against the age groups, smooth curves are obtained that are characteristic for the different types of cancer. Maxima are seen only with a few and relatively rare cancers, like testicular tumors, chorionepithelioma, and medulloblastoma. The majority of tumors show a steep increase of mortality with increasing age. While there is a slight preponderance of cancer mortality in females under 60, beyond this age cancer deaths in males becomes dominant.

Geriatric Gynecology at the New York Hospital

R. W. GAUSE. Obst. & Gynec. 5: 423-430, 1955.

A study was made of 1,171 gynecologic patients 65 and over admitted to the New York Hospital for the past decade. The geriatric patient is being admitted to the gynecology service in increasing numbers because of the great increase in the female population over 65. Vaginal repair operations have also increased greatly. About 1 out of every 4 hospital patients in this age group has a malignant disease. Advances in early diagnosis of cancer is less than in younger women because examinations in this group are too few.

Few patients are now denied gynecologic surgery because of age, and postoperative deaths in patients operated for benign disease are rare. Since use of cyclopropane and oxygen causes fewer depressions and other major complications, this anesthetic is given most commonly. Pentothal sodium is used widely for induction.

Blood is given when indicated and is never withheld because of age or cardiovascular status. All patients receive intravenous fluids, but in smaller amounts than younger women. Antibiotics are used freely, and early ambulation is encouraged. Use of anticoagulants is limited to patients with thrombophlebitis, and prophylactic anticoagulant therapy is given to individuals subjected to radical operations.

Physiologic depression should be avoided. The patient must be kept awake and active at all times, and the operation should never be carried to a state of profound depression.

Postoperative complications are common, since a large percentage of the patients have hypertensive or arteriosclerotic heart disease and many have coronary or vascular disease. Febrile urinary tract infection is one of the chief complications in spite of chemotherapy. Postoperative urinary difficulty occurs in half of the patients even though a catheter is inserted into the bladder at the time of operation and removed one to four days later.

Educational Approaches to Aging

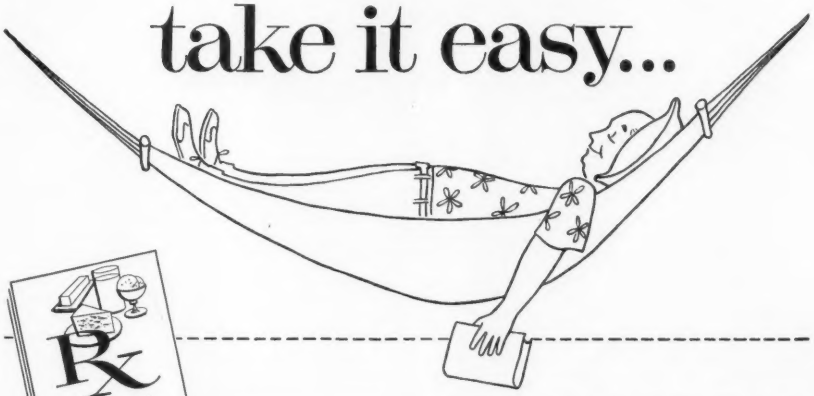
WILMA DONAHUE, PH. D. Merrill-Palmer Quarterly 1: 57-67, 1955.

Education for old age should begin early and should be aimed at all educational levels. Programs should include broader social living and citizenship as well as employment problems and leisure time activities. All groups concerned with older adults—such as business, industry, health agencies, education and welfare organizations—have a responsibility to work together toward better programs for elderly people.

In the employment field several types of approaches are being tested with good results: refresher courses and adult vocational education,

(Continued on page 44A)

when it's time to take it easy...



As energy requirements decrease, foods must be carefully chosen to provide nutrient needs. Medically supervised studies show that weight can be lost and desired weight maintained on a palatable diet of familiar and well-liked foods. This diet supplies generous quantities of high quality protein and calcium . . . nutrients frequently low in the food intake of older people.

The necessary foods for the diet can readily be chosen from the family table by the older person who lives with a younger family—are easily prepared by the person who cooks small meals.

The foods included in these diets provide all essential nutrients in amounts recommended for adults. Only calories are in deficit. Dairy foods are an important feature of these meals because of their high proportion of nutrients in relation to the calories they provide. Their taste appeal and variety make the diet easy to follow until the desired weight is lost.

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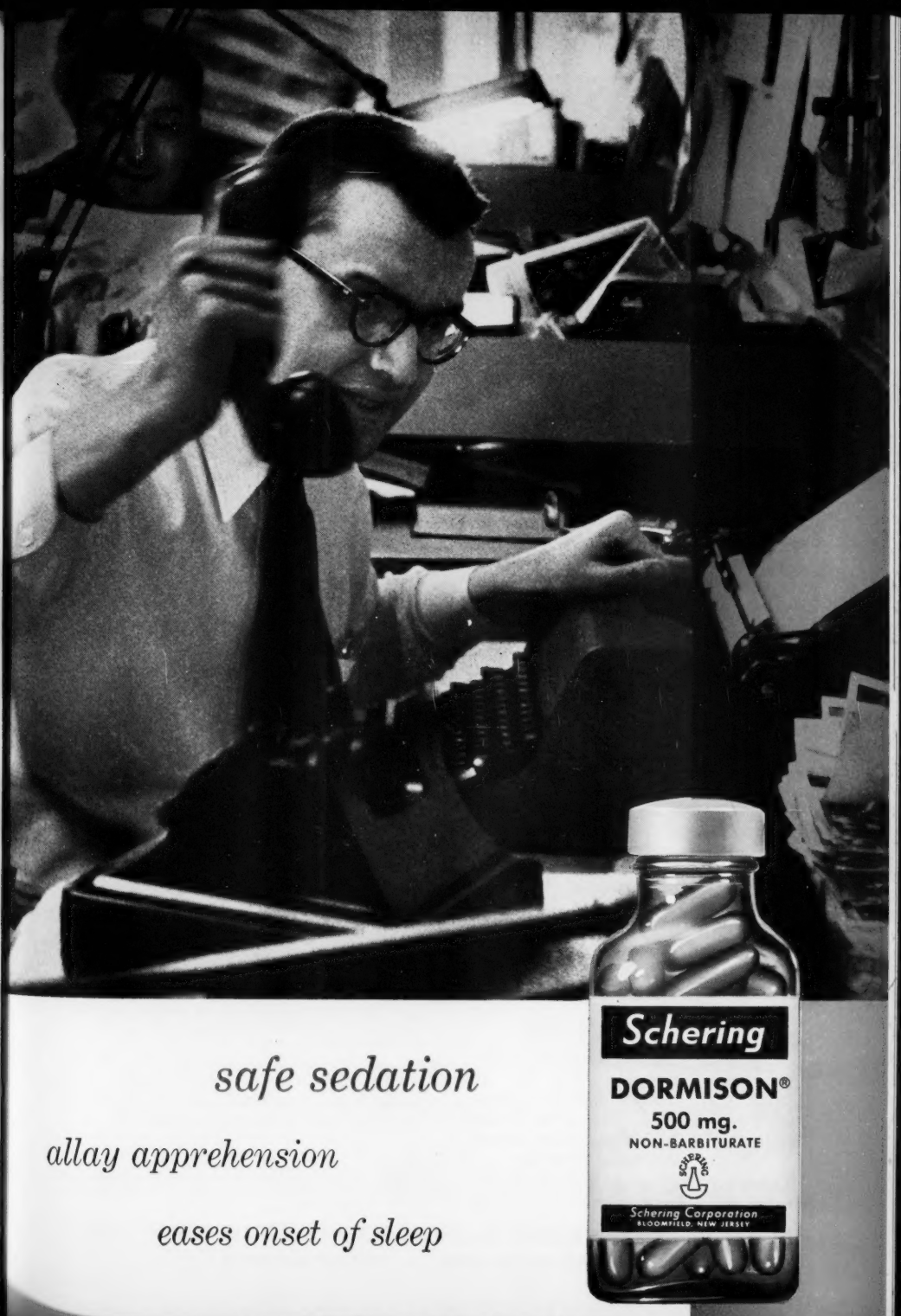

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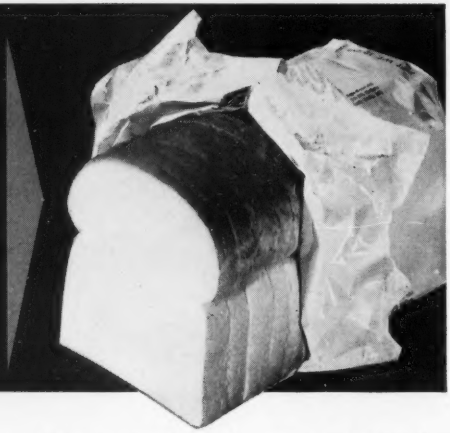
eases onset of sleep

Enriched Bread in *Dietary Planning*



BECAUSE of its nutritional, dietetic, and physiologic values, enriched bread simplifies in many ways the organization of dietaries suited to the special requirements of patients.

FOR THE SURGICAL PATIENT...

The first solid food after surgery is toasted enriched bread, slightly buttered. This practice has become a tradition—almost a ritual—because of the very nature of toast. It is bland, easily digested, and yields little inert residue. Its golden, warm appearance is pleasing to the eye; its mild taste appeals to the palate. Its nutrient energy plays a role in the physiologic and psychologic re-awakening of metabolic processes depressed under the "nothing by mouth" conditions immediately following surgery. With increasing tolerance for food it becomes an important component of the soft diet and later of the therapeutic diet.[1] Its valuable protein, B vitamins, iron, calcium and calories help the patient to regain nutritional efficiency.

FOR THE CONVALESCENT...

Enriched bread figures prominently in the dietary regimen in convalescence after acute infections, other serious illness, or trauma.

Supplying 13 grams of high grade protein per 5½ ounces (estimated average daily consumption), enriched bread makes an important contribution to the daily protein need. Its protein, comprising flour, milk, and yeast proteins, functions in the healing of wounds and in the rebuilding of wasted tissues.[2] In addition, 5½ ounces of enriched bread supplies on the average 0.37 mg. of thiamine, 0.23 mg. of riboflavin, 3.4 mg. of niacin, 4.1 mg. of iron, 137 mg. of calcium, and 418 calories.

FOR THE CHRONICALLY ILL...

In the formulation of palatable and nutritious menus for the debilitated, chronically ill, the advantages of enriched bread serve well.

In anorexia, enriched bread or toast stimulates the appetite. It is easily masticated and readily digested, features particularly important for elderly patients. Its favorable textural influence within the alimentary tract[3] promotes good utilization of ingested foods.

---

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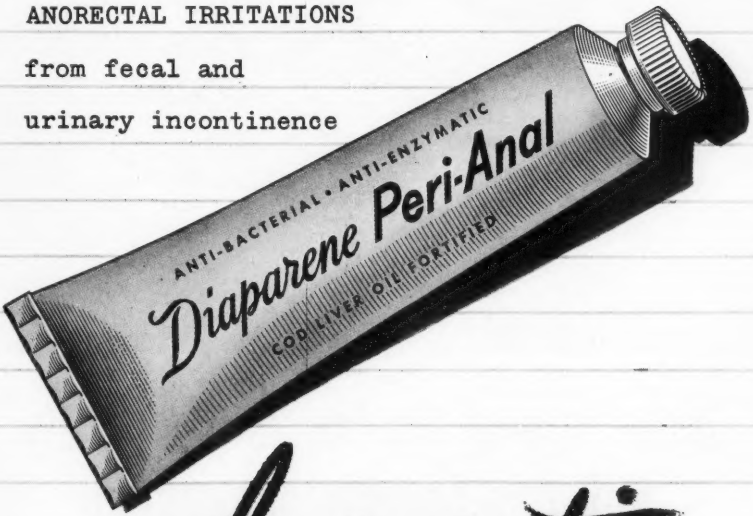

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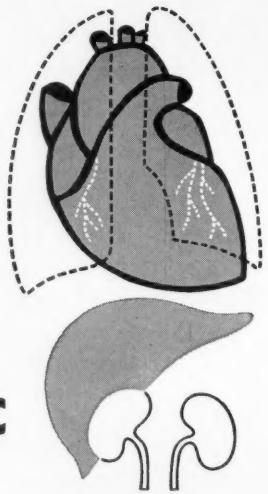

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for relief of aching, numbness, coldness, and blanching of the extremities due to vasospasm

PROPERTIES..... Ilidar relieves vasospasm and increases peripheral circulation by (1) direct vasodilation, and (2) adrenergic blockade, i.e., sympatholysis, adrenolysis, and epinephrine reversal.

INDICATIONS..... Peripheral diseases characterized by vasospasm, e.g., Raynaud's Disease, thromboangiitis obliterans, arteriosclerosis obliterans, endarteritis, postphlebitic syndrome, etc.

CONTRAINDICATIONS..... There are no known absolute contraindications. Use cautiously in the presence of asthma, coronary disease, cardiac decompensation, and peptic ulcer. Transient postural hypotension may result from overdosage.

DOSAGE..... One 25 mg tablet t.i.d., gradually increased as necessary (recommended maximum dosage, 300 mg daily).

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ILIDAR® *Phosphate—brand of azapetine phosphate (6-allyl-6,7-dihydro-5H-dibenz[c, e]azepine)*

Digests FROM CURRENT LITERATURE

(Continued from page 298)

senior achievement industries, counseling services offered by retired businessmen, employment bureaus, and arts and crafts training programs.

Preparation for the constructive use of leisure time is possible through institutes and conferences, library services, publications, lectures, leadership training, correspondence and extension courses, radio and film programs, and consultant and research services. Industrial programs for employees before retirement yield good results.

Professional people work with the aged in many fields: vocational counseling, personnel, community services, the ministry, adult education, recreation, medical and psychiatric services, geriatric nursing, old age homes, and occupational therapy. Although personnel training and courses in gerontology are often included in the regular programs of schools of public health, social work, medicine, nursing and education, the need for specialized curricula and intensive training programs is critical.

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An Effective Comprehensive Program for Geriatric Patients

M. B. FERDERBER, M. D., and G. P. HAMMILL. J.A.M.A. 157: 407-413, 1955.

The problems of geriatric patients in a public institution were met with a broad rehabilitation program and as a result, 23 per cent of the patients were discharged, 45 per cent became ambulatory, 12 per cent were subjectively improved, and 20 per cent showed no tangible improvement.

Patients bedridden for years were restored to a certain amount of normal activity by early ambulation, exercise in and out of bed, deformity correction, and recreation. With simple apparatus, convincing proof can be given the afflicted person that his physical reserve only needs developing to assist in his restoration.

Prolonged confinement to bed leads to incontinence, decubitus ulcers, emotional and morale problems, and undue hardship on nursing, dietary, and laundry personnel. The main purpose of exercise should be to restore evacuation and eliminate the bedpan. In the first four months of this program, linen replacements dropped 70 per cent.

Elderly patients generally have one of four conditions: cardiovascular disease, arthritis, amputation, or fractures. For hemiplegics, bedpan training is begun immediately on the return of consciousness. Amputees must be able to use crutches before being fitted with a prosthesis. Patients with arthritis or fractures do better if they get up and walk as soon as possible.

The patient is made a part of a rehabilitation team along with the physician, social worker, aide, and psychiatrist. The aide can be taught simple technics of bed exercises and movement to and from the bed. Liaison with the family during the patient's stay and after discharge is maintained by the social service department. Rehabilitation equipment is placed in the wards.

At the institution under discussion, a system of cooperation with private consultants and hospitals has been in operation for several years, with the county government paying for outside services. Existing public resources, such as the bureau of vocational rehabilitation, the Visiting Nurse Association, and volunteer groups, supplement the facilities.

A modern public institution for the chronically ill and aged should have (1) a complete floor devoted to rehabilitation, (2) a modified colony plan in the ambulant section, (3) a rolling terrain used to provide direct ground access for all patients, and (4) a central recreational center with multilevel auditorium and a chapel, as well as small recreational areas throughout the plant.

(Continued from page 46A)

FAT on the outside / THIN on the inside

The reduction of lean body mass . . . the withering away so commonly accepted with aging, represents an extensive loss of body protein.[1,2] This condition may also occur with an increase in the fat content of the body, so that old people, although obese, still may hide a considerable reduction in lean body mass.[3] Many times they look healthy and plump, but often are really suffering from reduction of lean body mass.

One excellent way to supplement the daily intake of protein is the low cost, easy to make, easy to take KNOX GELATINE DRINK. The *U.S.P., unflavored, all protein, sugarless* Gelatine Drink is *low in sodium,* non-allergenic and is in a neutral pH range. Only 28 calories to every 7 gram envelope. KNOX GELATINE has been recommended by doctors for over 64 years as a dietary supplement and source for readily available protein, from pediatrics to geriatrics.

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ALL PROTEIN / NO SUGAR

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Available at grocery stores in 4-envelope family size and 32-envelope economy size packages.

Charles B. Knox Gelatine Co., Inc., Johnstown, N. Y.

Digests FROM CURRENT LITERATURE

(Continued from page 44A)

Influence of Age and Vascular Disease on Cerebral Hemodynamics and Metabolism

J. F. FAZEKAS, J. FLEH, and F. A. FINNERTY. Am. J. Med. 18: 477-485, 1955.

Significant reductions in the rate of cerebral blood flow may occur without obviously impairing the activity of the central nervous system. In alert elderly patients, enough oxygen and other materials reach and are used by the brain cells even though cerebral blood flow is reduced. Moreover, the cells of the brain resist the aging process. Cases of early deterioration, then, may be primarily the result of vascular insufficiency, rather than of cell aging per se.

In elderly hypertensive patients, higher cerebral vascular resistance is found than in the normotensive elderly. The cause is probably degenerative vascular disease and increased blood vessel tone associated with the hypertension. The rate of cerebral blood flow or oxygen use does not differ between patients with or without hypertension in whom cerebral vascular insufficiency has not been detected, yet cerebral vascular insufficiency may occur earlier in patients with hypertension. These results suggest that hypertension is an accelerating factor in cerebral vascular disease, but not the cause.

A reduction of mean arterial pressure in older people with cerebral arteriosclerosis may result in transient cerebral ischemia. An acute reduction may lead to more cerebral ischemia in the rigid arteriosclerotic vessels because the available blood is shunted to relaxed vessels. In elderly hypertensive patients with cerebral arteriosclerosis even a gradual reduction of pressure may prove dangerous, even though the rate of total cerebral blood flow remains substantially the same.

The degree of arteriosclerotic involvement of the brain is not necessarily the same as elsewhere in the body. Cerebral blood flow and metabolism of patients with noticeable disease necessitating amputations, for example, may not differ from the control subjects.

The mental deterioration frequently associated with aging has been attributed to cerebral vascular insufficiency. A causal connection with brain deterioration has not been demonstrated, although both cerebral blood flow and metabolism are reduced in elderly patients with psychosis and cerebral arteriosclerosis.

Cerebral thrombosis in relatively young people is found with generalized cerebral vascular disease, indicating that the infarction may just be a more advanced degenerative process.

(Continued on page 48A)

Reduces elevated blood cholesterol levels.[1,2]

Improves hypercholesteremic patients with cardiovascular disease and angina and those with postoperative biliary dyskinesia, both subjectively and symptomatically.[1]

monichol*

Polysorbate 80, Choline, Inositol—the new physio-chemical complex)

normalizes cholesterol metabolism

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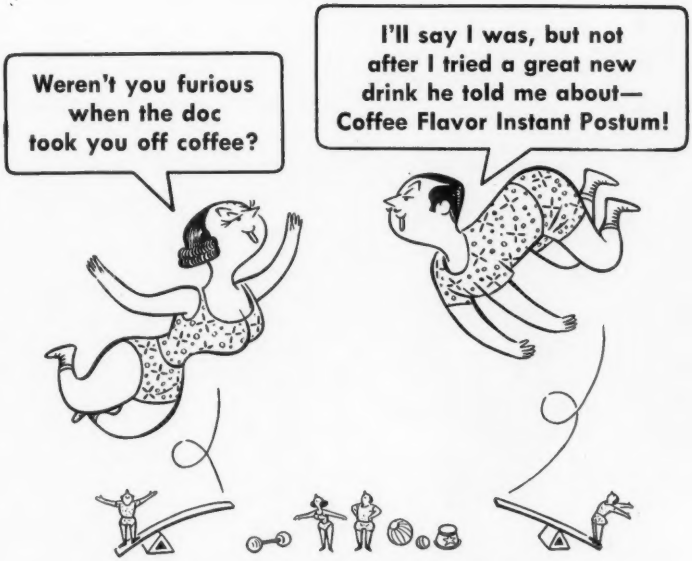
Literature available.

*Trademark

1. *Albert, A., and Albert, M.: Texas State J. Med. 50:814 (Dec.) 1954.*
2. *Sherber, D.A., and Levites, M.M.: J.A.M.A. 152:682 (June 20) 1953.*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I'll say I was, but not after I tried a great new drink he told me about— Coffee Flavor Instant Postum!

A patient's wrath can be quickly aroused by a "no coffee" edict. When this happens, a little Instant Postum advice may go a long way.

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Made of whole wheat and bran, the Instant Postum (Coffee Flavor or regular) in an average cupful contains only 10 mg. sodium and only 16 calories.

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Cincinnati 9, Ohio

"Manufacturers of high-speed passenger elevators for commercial buildings"

Digests FROM CURRENT LITERATURE

(Continued from page 46)

Mental Disorders of the Aged in Japan

N. SHINFUKU. Yonago Acta Medica 1: 115-123, 1954.

The rate of psychoses among the aged in Japan is far lower than in Europe or America. A survey of 60,587 psychiatric patients shows that after the age of 55 the relative incidence of psychoses decreases with increasing age.

It may be that many of the older psychotic patients are cared for in private households by their families. However, these findings result from the relatively low incidence of arteriosclerotic and senile psychoses. This is not due to fewer arteriosclerotic changes, since the death rate from cerebral hemorrhage in Japan is the highest in the world.

This suggests that only a few of arteriosclerotic patients present psychotic conditions, that the majority remain symptomless or unnoticed, and that in Japan it is easier for arteriosclerotics to adjust themselves without any significant mental disorders. The patriarchal family organization and ancestor worship make it easy for the aged to accomplish social adjustment. Also, the Buddhistic view of aging and death lessens the psychologic tension. This indicates that the aging process by itself does not produce mental disorders, but must be combined with other environmental or personality factors.

"prime" the patient with PRIMOPLEX parenteral!

All B vitamins, liver and C, packaged in individual patient doses.

For preventing and treating deficiencies of the B vitamins and ascorbic acid in persons of all ages. Each individual-dose package consists of a diluent and a powder, sufficient to prepare a single injection (2 cc.) containing:

Thiamine HCl (B_1)	10 mg.
Riboflavin (B_2)	10 mg.
Sodium Pantothenate	10 mg.
Niacinamide	50 mg.
Pyridoxine HCl (B_6)	5 mg.
Ascorbic Acid (C)	200 mg.
dl-Methionine	50 mg.
Vitamin B_{12}	15 mcgm.
Liver Injection Crude	2 mcgm.**
Choline Chloride	150 mg.
Folic Acid	3 mg.
Inositol	20 mg.

**VITAMIN B_{12} ACTIVITY EQUIVALENT TO 2 MCGM. CYANOCOBALAMIN

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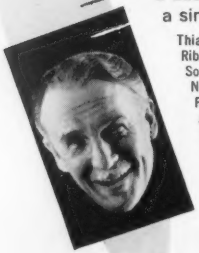

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Geriatric Liver and Vitamins Lederle

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Cogentin®

METHANESULFONATE (BENZTROPINE METHANESULFONATE, MERCK) (TROPINE BENZOHYDRYL ETHER METHANESULFONATE)

for maximal symptomatic relief in parkinsonism

MAJOR ADVANTAGES: Effective against rigidity.[1] Counteracts muscle spasm.[2] "Good against major tremor."[2]

Steadier hands for your palsied patient

For a single drug COGENTIN "is most often the best"[3] available for treating parkinsonism. It provides relief in *all* types—postencephalitic, idiopathic and arteriosclerotic forms of the disorder. Both young and old tolerate it.[2] In some cases it has been administered for months without ill effects.[1]

COGENTIN relieves even severe tremor.[2] Its strong antispasmodic action helps the patient to maintain an erect posture,[2] helps "counteract muscle spasm and frozen states."[2]

Dosage: Because of the prolonged action of COGENTIN effective relief may be obtained by a *single* 1 or 2 *mg.* dose taken orally at night. Each quarter-scored tablet contains 2 *mg.* COGENTIN.

References: 1. Neurology 2:233, 1952. 2. J.A.M.A. 156:680, 1954. 3. M. Clin. North America 38:485, 1954.

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GOOD FOR GRANDMA, TOO!

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A New Dietary Management for

CONSTIPATED ELDERLY

A bowel content modifier that softens dry, hard stools by dietary means without side effects.[1] Acts by promoting an abundant fermentative bacteria in the colon, thus producing soft, easily evacuated stools. Retards growth of putrefactive organisms. By maintaining a favorable intestinal flora, Malt Soup Extract provides *corrective therapy for the colon, too!*

DOSE: 2 tablespoonfuls b.i.d. until stools are soft (may take several days), then 1 or 2 Tbs. at bedtime.

*Specially processed malt extract neutralized with potassium carbonate. In 8 oz. and 16 oz. bottles.

1. Cass, L. J. and Frederik, W. S.: Malt Soup Extract as a Bowel Content Modifier in Geriatric Constipation. Journal-Lancet, 73:414 (Oct.) 1953.

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TREATMENT • SECURITY • COMFORT

for the aged patient

This modern, licensed private psychiatric hospital carries forward treatment designed to retard deterioration.

A high ratio of staff to patients and complete facilities allow indicated therapy and recreation on the basis of personal capacity.

Lovely country surroundings and separate accommodations for the elderly contribute to peace of mind.

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from rigidity to relaxation

When the rigidity and pain of arthritis and related rheumatoid disorders prevent the patient from enjoying a normal, satisfying life, Acetycol may open a road to rehabilitation. Therapy with Acetycol provides welcome relief of pain and increases the range of pain-free movement. Thus the patient is able to resume more normal activities in his work and relaxation.

The effectiveness of Acetycol is based on synergism between aspirin and para-aminobenzoic acid. The combination of these two agents produces high salicylate blood levels on relatively low dosages. Salicylated colchicine extends the effectiveness to cases of a gouty nature.

Acetycol also contains three essential vitamins often lacking in older patients: ascorbic acid, to prevent degenerative changes in connective tissue; thiamine and niacin, for carbohydrate utilization and relief of joint pain and edema.

Usual dosage—1 or 2 tablets three or four times a day.

Each Acetycol tablet contains:

Aspirin	325.0 mg.
Para-aminobenzoic acid	162.0 mg.
Colchicine, salicylated	0.25 mg.
Ascorbic acid	20.0 mg.
Thiamine hydrochloride	5.0 mg.
Niacin	15.0 mg.

Supplied: Bottles of 100 and 500.

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TRADEMARK

to relieve rheumatic pain

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make special diets more nutritious and better tasting

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Ovaltine in milk provides essential food elements that make it an ideal supplementary beverage with meals, for between meal snacks, and at bedtime.

Ovaltine in milk is easily digested and is delicious served either hot or cold. Its distinctive flavor appeals to both the young and the old.

Three servings daily (½ ounce of Ovaltine added to 8 fluid ounces of whole milk per serving) contain:

MINERALS		*Vitamin D	420 I.U.
*Calcium	1.12 Gm.	*Ascorbic acid	37.0 mg.
Phosphorus	940 mg.	*Thiamine	1.2 mg.
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Copper	0.7 mg.	Pyridoxine	0.5 mg.
Iodine	0.7 mg.	Vitamin B12	5.0 mcg.
Fluorine	0.5 mg.	Pantothenic acid	3.0 mg.
Cobalt	0.006 mg.	*Niacin	6.7 mg.
Sodium	560 mg.	Folic acid	0.05 mg.
Chlorine	900 mg.	Choline	200 mg.
Magnesium	120 mg.	Biotin	0.03 mg.
Manganese	0.4 mg.	*PROTEIN	32 Gm.
Potassium	1300 mg.	CARBOHYDRATE	65 Gm.
Zinc	2.6 mg.	FAT	30 Gm.
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*Vitamin A	3200 I.U.		

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Davol Inflatable Rubber Bed Pan is soft and flexible—an ideal appliance for patients who have difficulty using other types of bed pans. Rubber rim inflates to any degree desired—serves as a soft and comfortable cushion. Can be used for long periods without discomfort to the patient.

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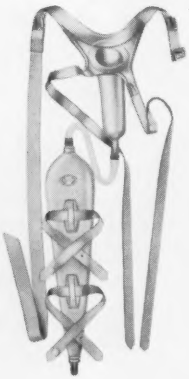
Cone-shaped sheath to prevent leakage. Adjustable waistbelt and elastic thigh straps. Oval, short bag, 14-oz. capacity.



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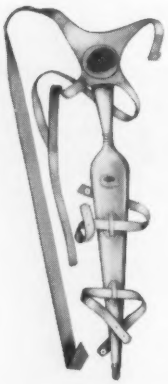
Amber latex top. Tapered latex sheath which may be cut to fit. Adjustable waistbelt and elastic thigh straps. Long bag, 20-oz. capacity. Over-all length, 24".



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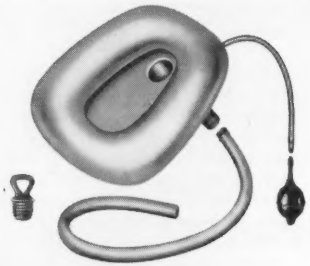
Cone-shaped latex sheath to prevent leakage. Adjustable waistbelt and elastic thigh straps. Long bag, 20-oz. capacity. Over-all length, 27½".



No. 842 Bed Pan *All-rubber, inflatable bed pan . . . made from soft, pure rubber, complete with inflating tube and deodorant cleansing soap.*

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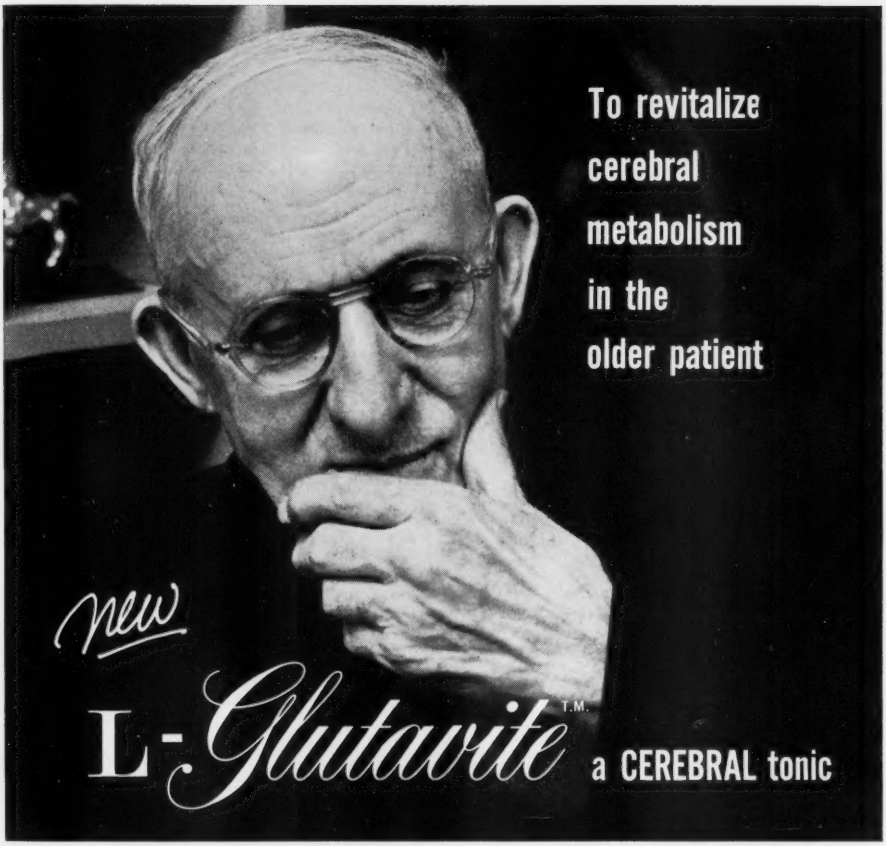


L-Glutavite is specifically formulated to assist in the treatment of geriatric patients whose symptoms of weakness, apathy and fatigue are characteristic of waning cerebral metabolism. Himwich[1] reports excellent results with sodium glutamate in such cases, while Lehmann states that "a trial with nicotinic acid is always indicated . . ."[2] By combining these key factors with other vital nutritional elements, L-Glutavite effectively increases cerebral blood flow while it enhances the ability of cerebral tissue to utilize the increased nutrients provided. This unique therapeutic formula helps the tired, the apathetic, the depressed individual to face life more actively, and with greater interest.

Supplied in cartons of 30 individual dosage packets; initial dose, 3-5 packets per day for 5 to 6 weeks. Pleasant-tasting appetite-stimulating powder, to be mixed in fruit juices or sprinkled on food. Contains monosodium L-glutamate, niacin, thiamine, riboflavin, ascorbic acid, ferrous sulfate and dicalcium phosphate, in high potencies.

1. Himwich H.E.: Paper presented at American Psychiatric Association meeting, St. Louis, May, 1954.
2. Lehmann, H.: 27th Annual Conference, Milbank Memorial Fund, New York, Paul B. Hoeber, Inc., 1952. p. 587.

GRAY PHARMACEUTICAL CO., INC., Newton 58, Massachusetts

Activities and Announcements . . .

Coming Meetings

The *University of Michigan* will hold its eighth annual conference on aging at Ann Arbor, on June 27 to 30, with the theme, "Aging—Applying Today's Knowledge Today." The conference program will consist of general sessions and workshops which will offer leaders, experienced workers, and newcomers to the field an opportunity to study the application of present day knowledge to the solutions to problems of older people in the fields of employment, housing, community organization, health, religion, education, use of leisure time, legislative action, and advanced planning. Further information can be obtained from the Division of Gerontology, 1510 Rackham, University of Michigan, Ann Arbor.

The *Ciba Foundation's* Second Colloquium on Aging will be held in London, England during the period of July 5 to 7. The subject of the Colloquium will be "Aging in Transient Tissues."

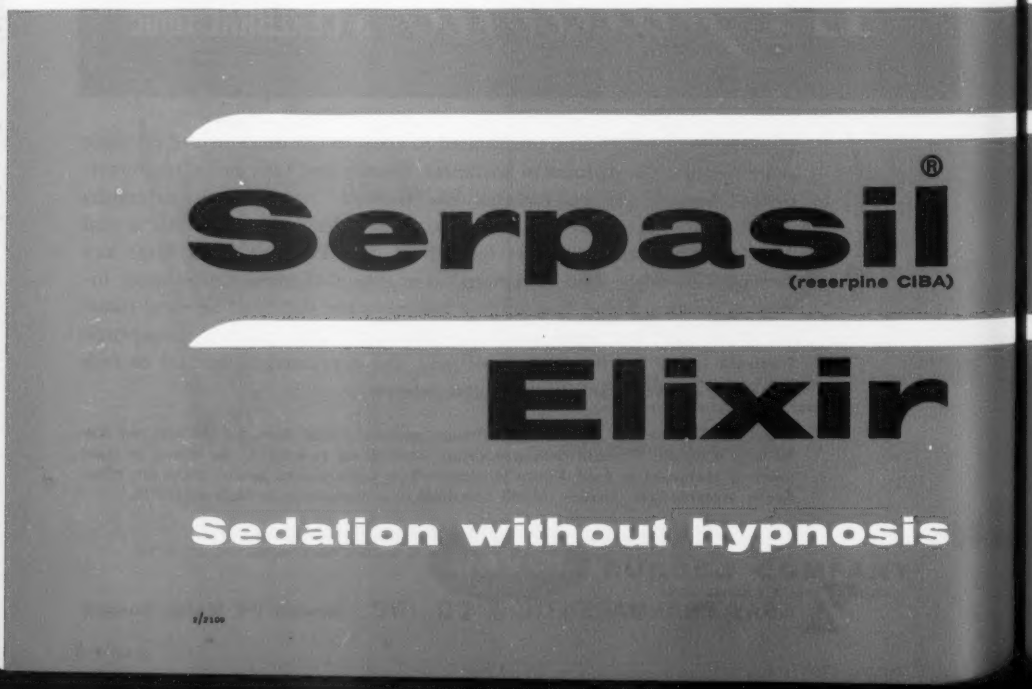
The 1955 subject of the Graduate Fortnight of the *New York Academy of Medicine* to be held October 10 to 21 will be "Problems of Aging." The program will consist of evening lectures, morning panel meetings, afternoon hospital clinics, a scientific exhibit, and other events. Additional information may be obtained from Dr. Robert L. Craig, New York Academy of Medicine, 2 E. 103 Street, New York 29, N.Y.

The 1955 annual meeting of the *Gerontological Society* will be held at the Sheraton Belvedere Hotel in Baltimore, Maryland, October 27 to 29. All those who wish to present papers should submit abstracts of 250 words or less in triplicate to Dr. Frederic D. Zeman, 111 East 88 Street, New York 28, N.Y.

•

Research Program Started

A three-year study into the question of what constitutes a "good old age" has been launched by the Community Service Society of New York. The investigation, which is aided by a $225,000 grant from the Rockefeller Brothers Fund, will be restricted to persons 60 years of age and over and will concentrate on the psychologic, social, and health problems associated with later life. The work will be done by a team of social scientists, social workers, and public health nurses, together with experts in the fields of psychiatry, medicine, and community planning.



Geriatric Center Opened

The Woodruff Center for the care of the chronically ill and infirm aged was opened in New Haven, Connecticut, with formal dedication ceremonies on April 14. In addition to complete medical, nursing, and social services for the 135 patients, there will be a well-equipped department of physical medicine and volunteer recreational program.

•

New Publications

An annotated bibliography on medical services relating to the aging has been prepared by the Committee on Indigent Care of the American Medical Association's Council on Medical Service. This listing contains references to books, pamphlets, and magazine articles, some of which are available to physicians on a loan basis from the Council. The material has been classified according to the various aspects of the aging problem – medical, socioeconomic, recreation, housing, and general. Copies are available from the Council.

"Day Centers for Older People" by Henry L. McCarthy and "Mobilizing Community Resources" by Jerome Kaplan are the newest pamphlets of a series of reports from public welfare departments on "How Public Welfare Serves Aging People." These booklets sell for 30 cents each and can be obtained from the American Public Welfare Association, 1313 East 60th Street, Chicago 37, Illinois.

Care of Chronic Disease

The chronically ill or disabled must be rehabilitated not only for best possible health but to conserve medical manpower and facilities. The Health Resources Advisory Committee of the Office of Defense Mobilization, Washington, D. C., has outlined a program under chairmanship of Dr. Howard A. Rusk. Recommendations are offered to medical and lay personnel in universities, hospitals, welfare and other community groups, and national associations.

•

Study of Aged in Norway

A comprehensive study, dealing with the diseases, economy, and employment opportunities of Norway's aged, was published recently. The report was prepared by a committee named in 1950 by the Norwegian Tuberculosis Association and the Norwegian Women's Health Association to examine every aspect of the problems of old age. The main recommendation calls for concerted efforts at the local level to consider the problems as a whole, with emphasis on more health and welfare centers for the aged.

Published as volume one in the series of Norwegian Gerontological Studies, the report will shortly be followed by another dealing with the health and living conditions of Norway's aged.

CIBA
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Especially indicated for Old People and Children

Highly compatible vehicle

New SERPASIL ELIXIR is compatible with Pyribenzamine® Elixir, dextro-amphetamine sulfate elixir, Antrenyl® Syrup, codeine phosphate, ephedrine sulfate, sodium salicylate and many other medications. Serpasil Elixir has a clear light-green color and a pleasant lemon-lime flavor. Each 4-ml. teaspoonful contains 0.2 mg. of Serpasil.

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particularly with the older patient

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Affords a Complete Formulary of Tar Products for the Management of

SENILE PRURITUS, ECZEMAS, SEBORRHEA and SIMILAR DERMATOLOGIC CONDITIONS

in pruritus "Tar baths are antipruritic in cases of generalized itching dermatoses and in generalized eczemas and psoriasis." ". . . this preparation [Alma-Tar Bath] provides one of the most effective and cleanest ways of preparing tar baths."

Sulzberger, M. B., and Wolf, J.: Dermatology: Essentials of Diagnosis and Treatment, Chicago, The Year Book Publishers, Inc., 1952, p. 42.

in seborrhea "Tar soap [or shampoo], rather than any oil shampoo, may be used after local medication."

Freeman, H. E.: South, M. J. 47:940 (Oct.) 1954.

ALMA-TAR BATH

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35% Oil of Cade by weight . . . in water-miscible base; 2 to 4 tbs. should be added to tub of water; patient should be immersed for 10 minutes with water and room at body temperature. Supplied in 8 fl. oz., qt., and gal.

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4% Oil of Cade in a bland base . . . may be applied topically when necessary to alleviate symptoms. Supplied 1 oz. tube, 4 oz. and 1 lb. jar.

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4% Oil of Cade in mild shampoo. Supplied 8 fl. oz., qt.

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5% Oil of Cade in sulfonated castor oil and water . . . indicated when soap is not desired. Supplied 8 fl. oz., qt.

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10% Oil of Cade in mild soap base. Supplied as 4 oz. cake.

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And where Crude Coal Tar Products are therapeutically preferable . . . The Almay Formulary Includes these Specific Products . . .

L.C.D. SOLUTION (Coal Tar Solution)

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Massengill Brings out Tablets Salcort

S. E. Massengill Company, Bristol, Tennessee, announces a new medication, Tablets Salcort, which utilizes the knowledge that cortisone and salicylates combined are more effective than either alone.

The medication gives prompt relief in rheumatic fever, rheumatoid arthritis, bursitis, Still's disease, and neuromuscular affections. It eliminates the side reactions of optimal cortisone or salicylate therapy, permitting effective therapy over a prolonged period. Salcort does not present withdrawal problems, as does cortisone when used alone.

The main ingredients, cortisone acetate and sodium salicylate, are complementary. Much lower cortisone-salicylate dosage is required, thereby markedly increasing the therapeutic efficacy.

Pad for Prevention of Bedsores

A new model of the Alternating Pressure Point Pad, used in the prevention and treatment of bedsores, has been announced by Air Mass, Inc. Cells of the pneumatic pad run the length of the mattress, allowing the edges of the pad to lie evenly without wrinkling. The pad reduces bedside nursing time in patient turning and massage and improves patient's comfort by automatically changing body pressure areas every four minutes. Pads are available through leading hospital equipment dealers. R. D. Grant Company, Cleveland, Ohio is the exclusive sales agency.

Continuous Release Tablet

The Stental Extentab, employing the new Robins Extentabs extended-action dosage form, contains 16.2 mg. phenobarbital in the outer coating and 32.4 mg. in the core. A single tablet provides the sedative effects of ¼ gr. phenobarbital, uniformly maintained over a ten to twelve-hour period.

Construction of the tablet, made by A. H. Robins Company Inc., Richmond, Virginia, assures therapeutic action promptly on ingestion without danger of "dumping" active ingredients at any time. One tablet administered morning and evening tranquilizes the patient throughout the day and night, freeing him from the usual strict q. 4 h. dosage schedule. Extentabs are applicable in the following conditions: anxiety tension states, insomnia, hypertension, angina pectoris, congestive heart failure, hyperthyroidism, vomiting of pregnancy, epilepsy, hyperirritability, restlessness and worry.

Clinical and biochemical consequences of

Vitamin Deficiencies

more serious in the aged[1]

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Allbee with C 'Robins' provides saturation dosage of the essential B vitamins, **plus 250 mg. vitamin C** —the highest ascorbic acid content of any water-soluble vitamin capsule.

1. Horwitt, M. K.: Jl. Am. Diet. Assn., 29:443, 1953.

. . . economical, too.

Allbee® *with* **C** Robins

Each capsule contains:

Thiamine hydrochloride	15 mg.
Riboflavin	10 mg.
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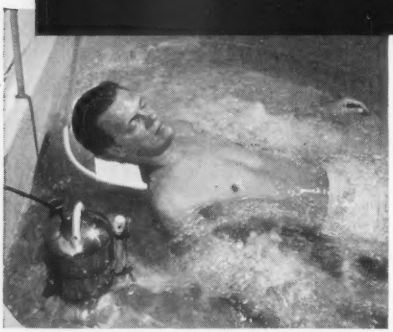
Nicotinamide	50 mg.
Ascorbic Acid	250 mg.

A. H. ROBINS CO., INC., RICHMOND 20, VIRGINIA

ETHICAL PHARMACEUTICALS OF MERIT SINCE 187

Overlooking Hydromassage?



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Are you aware that your patients with circulatory, muscular, bone and nervous disorders may now for the first time obtain the benefits of whirlpool hydromassage in the bathtub at home . . . and at reasonable cost?

HEAT PLUS MASSAGE

As you know, rehabilitation centers have used whirlpool therapy for years because of its unique ability to provide penetrating heat simultaneously with massage to stimulate circulation and loosen muscles and tendons. Unfortunately, its use has been greatly restricted because of the high cost for the equipment and its installation.

NOW A BATHTUB UNIT

The new portable Jacuzzi—which has exactly the same powerful action as conventional tank-mounted units—needs no special tank or plumbing. Consequently, cost is greatly reduced; and you can now prescribe whirlpool therapy for patients at home.

The Jacuzzi is made by one of the nation's foremost manufacturers of pumping equipment. It has been developed through seven years of research under supervision of physicians and is unconditionally approved for professional use by Underwriters Laboratories.

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chewing tablets

the NEWEST prescription for travel freedom from motion sickness

BONAMINE CHEWING TABLETS—the *only* motion-sickness medication which

(1) is pleasantly mint flavored, acceptable to children and adults who dislike taking pills
(2) is rapidly effective (most of the medication is extracted by 5 minutes of chewing)
(3) requires no water for administration
(4) promotes salivation and maintains the normal downward gastrointestinal gradient.

Well-tolerated BONAMINE is uniquely effective in a single daily dose. Notably free from side reactions.

BONAMINE medication is also indicated for the control of vertigo associated with vestibular and labyrinthine disturbances, cerebral arteriosclerosis, radiation therapy, Menière's syndrome and fenestration procedures.

BONAMINE CHEWING TABLETS contain 25 mg. of Bonamine each and are supplied in packets of 8, individually wrapped.

Also supplied as BONAMINE TABLETS of 25 mg. each, scored and tasteless, in boxes of 8 and bottles of 100 and 500.

*Trademark

Pfizer

PFIZER LABORATORIES, Brooklyn 6, N. Y.
Division, Chas. Pfizer & Co., Inc.

TO LEVEL
THE PEAKS
AND VALLEYS
OF YOUR
LABILE
HYPERTENSIVES

New
RAUVAL

RAUWOLFIA SERPENTINA TRADEMARK

Because RAUVAL contains *all* of the rauwolfia alkaloids, it provides a *natural* balance between hypotensive and sedative effects, and symptomatic relief is remarkably prompt.

This balance makes RAUVAL the drug of choice for patients with labile hypertension, especially when accompanied by tachycardia or neurosis.[1,2]

Supplied: Bottles of 100 and 1000 tablets in two strengths:
50 mg. s.c., red
100 mg. s.c., pink (double strength)

1. Wilkins, R. W.: Ann. Int. Med. 37:1144, Dec., 1952.
2. Wilkins, R. W., and Judson, W. E.: New England J. Med. 248:48, Jan. 8, 1953.

THE VALE CHEMICAL CO., INC.
pharmaceuticals
ALLENTOWN, PENNSYLVANIA

a valuable adjunct in the
PREVENTION
and **TREATMENT**
of **BEDSORES**

THE ALTERNATING
PRESSURE POINT PAD

In cases of threatened or existing decubitus ulcers (bedsores) the Alternating Pressure Point Pad has proved clinically helpful in 89% of cases. [1, 2, 3.]

Indicated in cases of long confinement, especially when turning of patient is not advisable, the Alternating Pressure Point Pad provides greater patient comfort, and reduces nursing care in half.

Nearly eight years of use in leading hospitals has proved the value of APP units in more than 25,000 cases.

1. Gardner, W. J., Anderson, R. M., and Lyden, Jr.; The Alternating Pressure Pad: An Aid to the Proper Handling of Decubitus Ulcers, Archives of Physical Medicine and Rehabilitation, (Sept.) 1954.
2. Gardner, W. J., and Anderson, R. M.: Alternating Pressure Alleviates Bed Sores. Mod. Hosp. 71:72 (Nov.) 1948.
3. Gardner, W. J.: Prevention and Treatment of Bedsores; Air Mattress Accomplishing Alternation of Pressure Points. J. A. M. A. 138:583 (Oct. 23), 1948.

Ask your hospital supply dealer or write direct to:

The R. D. GRANT Co.
805 Hippodrome Bldg.
Cleveland 14, Ohio

Manufactured by:
AIR MASS, Inc.
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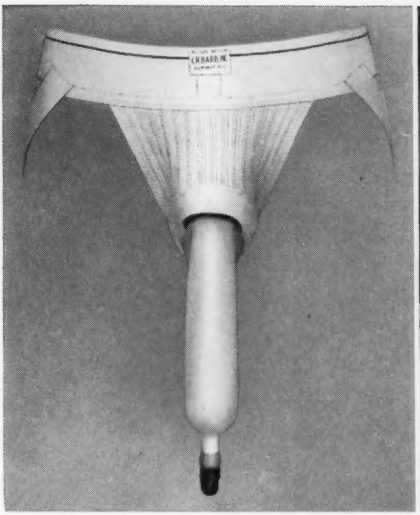

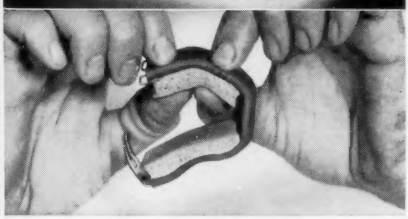

Male Enuresis Controlled By Collection or Occlusion

Comfortable McGuire Urinal Weighs only 3 ounces

PATIENTS welcome this new, McGuire Urinal because of its light weight and comfortable elastic belt and leg bands.

A conical penile sheath is easily cut to fit so there is no leakage even when sitting or lying down.

It is easily emptied at the bottom or attached to Bard leg bag or to bedside drainage tube.

The McGuire Urinal is available in 3 belt sizes—Small, Medium and Large. A urinal with half the capacity is available for boys.

Malleable Cunningham Clamp Easily Shaped to Fit

MANY thousands of patients have found the Cunningham Clamp effective and comfortable.

The rubber covered metal frame is easily shaped by the fingers to the proper contour. Further adjustment is possible with the ratchet catch which also guards against accidental opening.

Sponge rubber pads on top and bottom help to prevent undue restriction of blood vessels.

The Cunningham Clamp is available in four sizes—Infant, Juvenile, Regular and Large.

Available From Leading Surgical Supply Dealers

C. R. **BARD**, INC., SUMMIT, N. J.

for the "Sippy-diet" patient

a welcome *(and often necessary)* change from "milk-and-cream"

MULL-SOY® Powdered

Pioneer soy alternative to milk... reported to be "noticeably more soothing to the upper gastrointestinal tract and seemingly easier to digest."[1] Comparable to milk in buffering[2] and nutritional[3] qualities. Contains no cholesterol... and costs the patient *much* less than milk-and-cream. Easy to prepare—4 level tablespoonfuls to 8 oz. water. In 1-lb. tins at all drug outlets.

1. Balfour, D. C., Jr.: Am. J. Gastroenterol. 22:181, 1954. 2. Burke, J. O., et al.: Internat. Rec. Med. & Gen. Practice Clin. 167:587, 1954. 3. Sternberg, S. D., and Greenblatt, I. J.: Ann. Allergy 9:190, 1951.

Are you wondering how MULL-SOY Powdered tastes? Return this coupon for professional trial samples and see for yourself how *pleasant* it can be for your milk-weary or milk-intolerant ulcer patients.

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Prescription Products Division, Dept. 201
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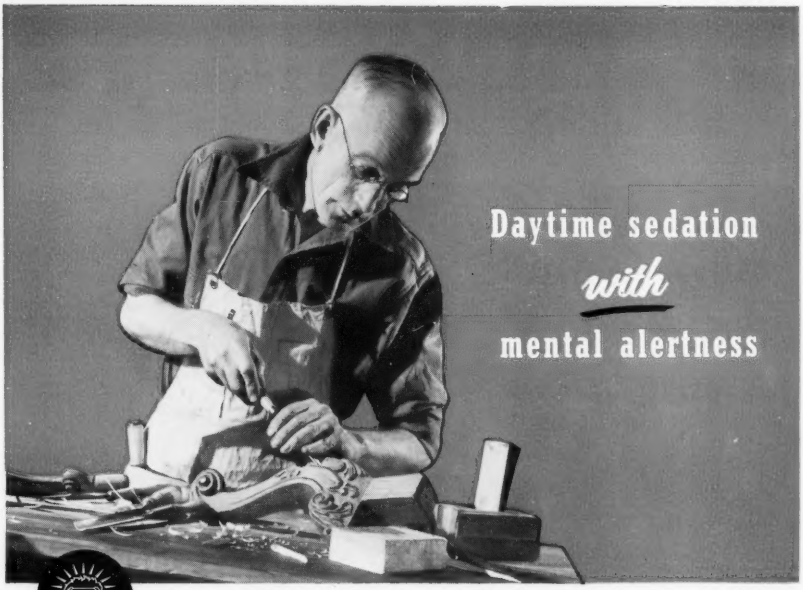

MEBARAL®

BRAND OF MEPHOBARBITAL

—in depressed and agitated states

psychoneurosis
hypertension
hyperthyroidism
convulsive disorders
difficult menopause
hyperhidrosis

Neurotic depression hiding beneath the disguise of multiple physical complaints is an everyday problem in medical practice.

For effective sedation in these cases, and as a means of restoring harmonious relations between patient and environment, Mebaral has been found especially suitable because it lacks excessive hypnotic action.

DOSAGE:

Adults—32 mg. to 0.1 Gm. (optimal 50 mg.), 3 or 4 times daily.

Children—16 to 32 mg., 3 or 4 times daily.

SUPPLIED:

Tablets of 32 mg. (½ grain)
50 mg. (¾ grain)
0.1 Gm. (1½ grains)
0.2 Gm. (3 grains) scored

Mebaral, trademark reg. U. S. & Canada

WINTHROP-STEARNS INC. New York 18, N. Y. • Windsor, Ont.

"Premarin" with Methyltestosterone effectively suppressed postpartum breast engorgement in 96.2 per cent of a series of 267 patients.[1]

"Premarin" with Methyltestosterone successfully prevents postpartum breast engorgement and suppresses lactation with virtually none of the side effects referable to either estrogen or androgen employed alone.

None of the 267 patients treated with "Premarin" with Methyltestosterone experienced nausea, vomiting, breast abscess, excessive lochia, withdrawal bleeding, or virilization.[1]

In addition, patients in this series were singularly free from the mental depression which usually appears on the fourth or fifth day of the puerperium.[1a]

Suggested Dosages: Some clinicians recommend intensive short duration therapy while others advocate lower dosage levels extended over a longer period of time. In either case, it is important to start therapy as soon as possible after delivery. Details on *short duration* therapy (one week) and *step-down* therapy (10-15 days) will be supplied on request.

1. Fiskio, P. W.: To be published; 1a. Personal communication.

"PREMARIN"® with METHYLTESTOSTERONE

ideal preparation for combined estrogen-androgen therapy

Supplied in two potencies: *Yellow* tablets (No. 879) contain 1.25 mg. of conjugated estrogens (equine) and 10 mg. of methyltestosterone; *red* tablets (No. 878) contain 0.625 mg. and 5 mg. respectively. Available in bottles of 100 and 1,000.

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